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A MANUAL

OF THE

PRACTICE OF MEDICINE

PREPARED

ESPECIALLY FOR STUDENTS

BY

A. A. STEVENS, A.M., M.D.

PROFESSOR OF THERAPEUTICS AND CLINICAL MEDICINE IN THE WOMAN'S MEDICAL COLLEGE OF PENNSYLVANIA; LECTURER ON PHYSICAL DIAGNOSIS IN THE UNIVERSITY OF PENNSYLVANIA; PHYSICIAN TO THE EPISCOPAL HOSPITAL AND TO ST. AGNES'S HOSPITAL; ASSISTANT PHYSICIAN TO THE PHILADELPHIA HOSPITAL; FELLOW OF THE COLLEGE OF PHYSICIANS OF PHILADELPHIA, etc.

> ——"is an arch where through
> Gleams that untravelled world whose margin fades
> Forever and forever as we move."

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PREFACE TO THE EIGHTH EDITION.

In the preparation of a new edition of this Manual the author's endeavor has been to bring the entire contents up to date. Not only has the text been thoroughly revised, but much new material has been introduced, and many articles, especially in the section dealing with Diseases of the Nervous System, have been rewritten.

It is hoped that in its present form the work may still be considered as affording a concise, but clear and accurate, representation of the essential facts of the Practice of Medicine.

A. A. S.

96383

PREFACE TO THE FIRST EDITION.

POPE says, " Half our knowledge we must snatch, not take." If this be true of general knowledge, it is certainly true of the knowledge of medicine as it is taught in the schools of to-day. In view of this fact, there seems to be a real need for books which present their subjects in an assimilable form.

At the request of many students the author has written this book with the hope that it may serve as an outline of Practice of Medicine, which shall be enlarged upon by diligent attendance upon lectures and critical observation at the bedside.

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A MANUAL

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PRACTICE OF MEDICINE

DISEASES

OF THE

DIGESTIVE SYSTEM.

THE TEETH AND GUMS. l, 7

Delayed dentition and the eruption of badly formed teeth may result from rickets or congenital syphilis.

Caries of the teeth results from many conditions, notably an unnatural softness of the teeth, lack of cleanliness, the use of certain drugs, dyspepsia, and diabetes.

Hutchinson's Teeth.—The lateral incisors of the upper jaw are pegged, and the central incisors of the same jaw have convex sides and crescentic notches on their cutting-edges. These peculiarities indicate hereditary syphilis, and are noted only in the permanent teeth.

A blue line on the gums near the insertion of the teeth usually indicates chronic lead-poisoning. Copper- and silver-poisoning occasionally produce similar lines.

Spongy, bleeding gums are often associated with scurvy. Swelling of the gums, with tenderness and salivation, is indicative of mercurial poisoning (ptyalism).

THE TONGUE. L1'/

Fur on the Tongue.—This consists for the most part of accumulated epithelial cells, particles of food, and micro-organisms, and occurs in a great variety of diseases, both local and general.

A light, uniform coat is often noted in health, particularly in those who sleep with the mouth open. Other causal conditions are: (1) Febrile diseases. (2) Dyspepsia. (3) Catarrhal conditions of the nose and throat. Very little diagnostic significance can be attached to the appearance of the tongue in diseases of the stomach.

Circumscribed furring often indicates local disturbance, as a jagged tooth or tonsillitis.

Unilateral furring may result from disturbed innervation, as in conditions affecting the second and third branches of the fifth nerve. It has been noted in neuralgia of those branches, and in fractures of the skull involving the foramen rotundum.

The dry, brown and *fissured tongue* is noted in low fevers, as typhoid fever, typhoid pneumonia, typhoid dysentery.

A red, beefy tongue is noted in chronic wasting diseases. It is of quite frequent occurrence in dysentery and in diabetes.

The "strawberry tongue" is characterized by a white fur, through which project bright-red and prominent papillæ. It is seen in the early stage of scarlet fever.

DISCOLORATION OF THE TONGUE.

Black Tongue (Nigrities).—This is a parasitic affection of the tongue, characterized by the appearance of black patches on the center of the dorsum, with great prolongation of the filliform papillæ.

Bluish-black discoloration of the tongue is observed in Addison's disease.

Leukoplakia Buccalis.—In this condition there are slightly elevated, smooth, opaque, whitish plaques on the lingual or buccal mucous membrane. There are no subjective symptoms. Excessive smoking is a common cause. Syphilis appears to be a factor in some cases.

TREMOR OF THE TONGUE.

Trembling of the tongue is noted in many conditions; it is particularly marked in low fevers (typhoid), in alcoholism, in paretic dementia.

FISSURES ON THE TONGUE.

Fissures on the tongue may result from severe glossitis, syphilis, carcinoma, tuberculosis, or the impact of a jagged tooth.

SCARS ON THE TONGUE.

Scars on the tongue often result from syphilitic lesions or from the tooth wounds of epilepsy.

FETOR OF THE BREATH.

This is often due to local inflammation, as chronic rhinitis, tonsillitis, etc.; to the retention of decomposing food, to caries of the teeth, to certain lung diseases, especially gangrene and bronchiectasis, to dyspepsia, and to the ingestion of certain foods or drugs.

THE APPETITE.

Bulimia, or *inordinate appetite*, is a common symptom in nervous dyspepsia, hysteria, diabetes, and in certain insanities, notably in paretic dementia. It may be due to intestinal parasites.

Anorexia, or *loss of appetite*, is a symptom common to many conditions.

Pica is a craving for unnatural articles of food, and is noted particularly in chlorosis, insanity, and pregnancy.

DYSPHAGIA.

Dysphagia, or difficult swallowing, may result from: (1) Local inflammation, especially tuberculous ulceration of the throat or larynx. (2) Stricture due to the healing of an ulcer (corrosive poisons, syphilis, typhoid fever). (3) Cancer of the esophagus. (4) Spasm of the esophagus (hysteria). (5) A foreign body. (6) Pressure on the esophagus (aneurysm, mediastinal tumor, enlarged glands, pericardial effusion). (7) Paralysis, local, as in diphtheric paralysis; or centric, as in bulbar disease.

VOMITING, OR EMESIS.

Etiology.—(1) Toxic, from ptomains, drugs, uremia, and the specific fevers. (2) Centric disease, as cerebral tumors and meningitis; this type is often unaccompanied with nausea, and does not relieve the associated headache. (3) Diseases of the stomach, as ulcer, cancer, dilatation, catarrh, etc. (4) Reflex, as from pregnancy, uterine or ovarian disease, irritation of the fauces, worms, biliary colic, etc. (5) Intestinal obstruction: this is often fecal. (6) Disturbed cerebral circulation, as in swinging and in sea-sickness. (7) Certain nervous affections, as hysteria, migraine. (8) Periodic vomiting may be in itself a neurosis, or may be associated with the gastric crises of locomotor ataxia. (9) Esophageal vomiting results from obstruction, and the vomit is alkaline in reaction.

THE VOMIT.

Watery or mucous vomit is noted in chronic gastritis, in certain forms of nervous dyspepsia, in cerebral disease, and after persistent emesis, as in cholera.

Bilious or green vomit is not diagnostic of any special condition; it may occur in any case in which there are persistent vomiting and retching.

Bloody Vomit (Hematemesis).—For causes, see page 62. When present in large amount, the blood can usually be recognized by the unaided eye; small amounts may be detected by the microscope, by the spectroscope, or by chemical tests.

Test for Blood.—Evaporate some of the filtered coffee-grounds vomit in a watch-glass, scrape off some of the dried material; add a trace of finely pulverized salt; place the mixture on an object-glass, and cover. Allow one or two drops of glacial acetic acid to run under, and again evaporate; when dry, allow one or two drops of distilled water to flow under to dissolve the crystals of salt. Under the microscope brown rhombic crystals of hematin appear.

Purulent vomit may result from the rupture of an abscess into the esophagus or stomach or from phlegmonous gastritis.

Fecal vomit (*stercoraceous vomit*) indicates intestinal obstruction or a gastro-colic fistula, the result of ulcer or cancer. It is recognized by its odor and appearance.

Profuse Vomit.—The ejection of large quantities of frothy fermented material is highly significant of gastric dilatation.

Vomiting without nausea, distress, or other gastric phenomena occurs in certain neuroses of the stomach, in hysteria, uremia, and in brain disease, as tumor or as a precursor of apoplexy.

EXAMINATION OF THE GASTRIC CONTENTS.

The *test-breakfast* of Ewald and Boas consists of a roll and from 10 to 14 fluidounces of water or weak tea. It is given in the morning on an empty stomach, and is removed in one hour by aspiration or expression. The roll should be thoroughly masticated. This breakfast affords the most satisfactory means of determining the secretory activity of the stomach. Riegel's *test-meal*, however, is better adapted to determining the total functional activity of the stomach. It consists of a plate of meat-broth, a beef-steak weighing from 5 to 7 ounces, 1½ ounces of mashed potatoes, and a roll. The contents are removed in three or four hours after the ingestion of the meal.

Test for Free Acids.—Filter-paper soaked in a solution of Congo-red and dried turns blue in the presence of free acids. A saturated alcoholic solution of tropeolin 00 turns from a brownish yellow to a dark brown when brought in contact with fluids containing free acids.

Qualitative Tests for HCl.—Günzburg's phloroglucin-vanillin test will react with 1 part of HCl in 15,000 parts of water. The solution consists of 2 parts of phloroglucin, 1 part of vanillin, and 30 parts of absolute alcohol. When a few drops of this solution are heated with an equal quantity of the filtrate contained in a porcelain dish, a beautiful red color appears at the margin of the fluid. Boas states that the test is still more delicate when 100 parts of 80 per cent. alcohol are substituted for the 30 parts of absolute alcohol.

Boas' resorcin-sugar test gives a similar reaction. The reagent consists of 5 parts of resorcin, 3 parts of sugar, and 100 parts of diluted alcohol.

Total Acidity.—This is determined by allowing a decinormal alkali solution (water, 10 c.c.; potassium hydrate, 56 mg.) to flow from a buret, drop by drop, into a beaker containing 10 c.c. of filtered gastric juice, to which have been added as an indicator two drops of a 1 per cent. alcoholic solution of phenolphthalein. The test is completed when the red color produced no longer disappears on shaking the solution. Ten c.c. of normal gastric juice usually require from 4 to 6.5 c.c. of the standard alkali solution.

Since 1 c.c. of the alkali solution is equivalent to 0.00364 gram of HCl, it follows that the percentage of the latter in a given specimen will equal the number of cubic centimeters of the alkali solution required multiplied by 10, and again by 0.00364.

Quantitative Test for Free Hydrochloric Acid.—*Mintz's Color Method.*—To 10 c.c. of the filtrate add a decinormal solution of sodium hydrate from a buret until a droplet (removed with a platinum loop) of the fluid no longer reacts with Günzburg's reagent. The number of cubic centimeters of the alkaline solution used, multiplied by 10 and then by 0.00364, gives the percentage of free hydrochloric acid. This method, which is sufficiently accurate for clinical purposes, is based upon the supposition that the alkali first unites with the free acid before it affects the acid in organic combinations.

Test for Lactic Acid.—The presence of lactic acid in the stomach-contents simply indicates the existence of subacidity and of stagnation. These two conditions are never so constantly present nor so intense as in carcinoma (Riegel). When free HCl is present in sufficient quantities, it is unnecessary to test for lactic acid.

Uffelmann recommends a mixture of 10 c.c. of a 4 per cent. carbolic acid solution and 20 c.c. of distilled water, to which is added one drop of the official liquor ferri chloridi. This makes a clear amethyst-blue solution. The reagent must always be prepared at the time of making the test.

If the solution turns yellowish green on the addition of filtered gastric contents, the presence of lactic acid is demonstrated. As the blue serves only as a contrast color, a very dilute solution of iron chlorid alone (one drop of liquor ferri chloridi in 50 c.c. of distilled water) suffices. As other substances, such as sugar, alcohol, acid phosphates, etc., give a somewhat similar reaction, the test is made more reliable by exhausting the gastric filtrate with pure ether (10 vol.), evaporating the ether, and adding the reagent to an aqueous solution of the residue.

Test for Acetic Acid.—This acid may be detected by its odor. The production of a blood-red color on the addition of a neutral solution of ferric chlorid to an aqueous solution of the ethereal extract, which has been neutralized with sodium carbonate, also indicates the presence of acetic acid.

Test for Butyric Acid.—This acid strikes a brownish-yellow color with Uffelmann's reagent. Its odor is also characteristic.

Tests for Rennet and Rennet Zymogen.—**Rennet.**—Add 5 c.c. of filtrate, which has been exactly neutralized with a decinormal alkaline solution, to an equal quantity of neutral raw or boiled milk, and keep the mixture at a temperature of about 100° F. If a flocculent coagulate forms in from ten to thirty minutes, the presence of rennet is revealed.

Rennet Zymogen.—Render 5 c.c. of filtrate very slightly alkaline with a 1 per cent. solution of sodium bicarbonate, add about 2 c.c. of a 1 per cent. solution of calcium chlorid, then mix with an equal quantity of pure milk, and keep the mixture at a temperature of 100° F. If zymogen is present, coagulation occurs in the usual time.

Test for Pepsin.—If free hydrochloric acid is present, the presence of pepsin in sufficient quantities may be assumed. To determine the presence of pepsin, pour 10 c.c. of filtrate into a test-tube. If free HCl is absent, add a sufficient quantity of acid to cause the appearance of the Congo reaction. Drop discs (1.5 mm. thick and 10 mm. in diameter) of hard-boiled egg into the mixture, and put the test-tube into the thermostat at 100° F. If sufficient pepsin is present, the discs will be completely dissolved in from one-half to one hour.

Test for Carbohydrates.—When starch digestion is arrested too early, as in cases of excessive secretion of HCl, Lugol's solution gives a blue or purple coloration with the gastric contents. Complete absence of color reaction indicates very active starch digestion (subacidity).

The Absorptive Power of the Stomach.—This is usually determined by the time required for free iodin to appear in the saliva after the ingestion of potassium iodid. The saliva is received on filter-paper, impregnated with starch, a drop or two of fuming nitric acid is then added, and the appearance of a blue color proclaims the presence of iodin. Normally the saliva should yield the reaction for iodin in from ten to fifteen minutes after the ingestion of a capsule containing 0.1 gram of potassium iodid. Care must be taken that none of the drug adheres to the outside of the capsule. This test cannot be regarded as being very reliable.

The Motor Power of the Stomach.—Ewald has suggested the use of salol, which escapes from the stomach into the intestine, where it is broken up into salicylic acid and phenol. Normally salicyluric acid appears in the urine in from forty to seventy-five minutes after the ingestion of 1 gram of salol. Filter-paper moistened with urine containing salicyluric acid assumes a violet color when treated with a 10 per cent. ferric chlorid solution.

Riegel's test is more reliable. If it is found that seven hours after a test-meal of broth, beef-steak, mashed potatoes, and a roll (see p. 21) much food is still left in the stomach, the motor power is reduced.

No remains of the *test-breakfast* should be found after two hours.

If much water is recovered in one and one-half hours after the ingestion of 500 c.c. of cool water, there is motor insufficiency, probably the result of muscular weakness, and not retention from pyloric obstruction.

ACIDITY OF THE GASTRIC CONTENTS.

Normal acidity is due to hydrochloric acid, but other acids are frequently formed during the digestive process, such as lactic, butyric, and acetic acids. The quantity of hydrochloric acid in normal gastric juice varies from 0.14 to 0.2 per cent., more acid being secreted after a heavy meal than after a light one.

Hyperacidity (**hyperchlorhydria**) results from a variety of causes. Early life, the nervous temperament, mental overexertion, and the persistent use of highly seasoned foods are general predisposing factors. It is frequently present in neurasthenia and in hysteria. It may attend the gastric crises of locomotor ataxia. It may result from the abuse of tobacco. It is present, as a rule, in ulcer of the stomach. It is a common symptom in chlorosis. It sometimes occurs in cholelithiasis and in nephrolithiasis.

Subacidity and Anacidity (**Hypochylia Gastrica and Achylia Gastrica**).—Decreased secretion of gastric juice is seen in chronic gastritis; in gastric cancer; in atrophy of the gastric tubules; in passive congestion of the stomach; often in febrile diseases; often in severe anemia; and in certain neuroses, as neurasthenia, hysteria, and some forms of nervous dyspepsia.

RUMINATION, OR MERYCISMUS.

Rumination is a condition, rarely observed in man, in which the food is regurgitated from the stomach and subjected to a second mastication. It is the result of a neurosis, and is generally found in association with hysteria, epilepsy, neurasthenia, or idiocy. It is sometimes hereditary or acquired by imitation.

HICCUP.

Hiccup, or singultus, results from a clonic spasm of the diaphragm, and is often noted as a temporary condition after eating or drinking. Persistent hiccup is sometimes present in extreme exhaustion following acute or chronic diseases. It may also result from irritation of the phrenic nerve, as from the pressure of a thoracic aneurysm. It may be reflex from stomachic, hepatic, intestinal, or peritoneal disease. It may be due to hysteria.

ABDOMINAL PAIN AND TENDERNESS.

Diffuse abdominal tenderness is noted in peritonitis, in hysteria, and in rheumatism of the abdominal muscles.

Persistent abdominal pain results from the various visceral diseases, chronic peritonitis, abdominal aneurysm, and disease of the spinal vertebræ.

Colic is a painful spasm of a mucous canal. The chief varieties are biliary, intestinal, renal, and pancreatic.

Painful defecation results from constipation, anal fissure, dysentery, piles, ulceration, stricture, prolapse of the rectum, and inflammatory conditions of neighboring organs, as the uterus or prostate gland.

THE STOOLS.

Blood in the Stools (*Entrorrhagia or Melena*).—The blood is nearly normal in appearance after profuse hemorrhages, or when it has been quickly discharged, as in piles and fissure. Retained blood imparts a black or tarry appearance to the stools.

Melena results from: (1) Traumatism; (2) acute inflammation of the bowels, as in enteritis and dysentery; (3) passive congestion, as in chronic heart and liver disease; (4) vicarious menstruation (extremely rare); (5) blood dyscrasia, as in scurvy, purpura, infectious fevers, etc.; (6) rupture of an aneurysm; (7) ulcers in the intestines, as simple duodenal ulcers, typhoid, dysenteric, tubercular, or malignant ulcers; (8) intussusception; (9) the passage of blood from the stomach in hematemesis; (10) hemorrhagic infarction of the bowel from embolism or thrombosis of the mesenteric artery; (11) piles, fissure, fistula.

Watery or serous stools are noted in choleraic diseases, in nervous diarrhea, in the colliquative diarrhea which terminates wasting diseases, in severe enteritis, and in corrosive poisoning, as by arsenic or antimony.

Green stools may result from an excessive amount of bile. They are also common in the diarrheas of young children, as a result of the abnormal decomposition of the bile pigment.

Black stools follow intestinal hemorrhage and the use of certain drugs, as charcoal, bismuth, iron, tannin, etc.

Red stools usually indicate blood, but they may be tinged red after the administration of hematoxylin (logwood).

Mucous stools are noted in intestinal catarrh, particularly when the lower bowel is affected, as in enterocolitis and dysentery.

Fatty stools result from the ingestion of large quantities of fats, from the absence of bile, and from chronic pancreatic diseases.

Purulent stools result from fistula in ano, dysenteric, syphilitic, or malignant ulceration, or the rupture of abscesses into the bowel, as prostatic and pelvic abscesses.

Lienteric stools, those which contain much undigested food, are noted in inflammatory conditions of the stomach and upper bowel.

ABDOMINAL DISTENTION.

Causes.—(1) Enlargement of the various organs from tumors or other causes. Recognized by the history, irregular enlargement, and special symptoms referable to the organ affected. (2) Ascites. Recognized by movable dulness with superincumbent tympany, and fluctuation. (3) Chronic peritonitis (tuberculous or cancerous) with effusion. Recognized by the history, progressive emaciation, presence of a primary lesion elsewhere, and detection of tumor-like masses, with, perhaps, pain and tenderness. (4) Tympanites. Recognized by universal tympany on percussion. (5) Pregnancy. Recognized by suppression of menses, morning emesis, pigmentation of mammary areola, softening of the cervix, intermittent uterine contractions, etc. (6) Distention of the bladder. Recognized by the history, location of dulness, and results of catheterization.

STOMATITIS.

Definition.—Inflammation of the mouth.

Etiology.—(1) Mechanical, chemical, thermal, or parasitic irritation. (2) Mercurial poisoning. (3) Cachectic states, as in phthisis, cancer, and diabetes. (4) It is most commonly seen in young children in association with gastro-intestinal disturbances, brought about by artificial feeding, warm weather, and bad hygienic surroundings.

Varieties.—(1) Catarrhal. (2) Aphthous. (3) Ulcerative. (4) Parasitic (thrush). (5) Gangrenous. (6) Mercurial.

General Symptoms.—Heat and pain in the mouth, increased flow of saliva, fetor of the breath, restlessness, languor, disinclination to nurse, and perhaps some fever.

CATARRHAL STOMATITIS.

(Simple Stomatitis.)

Symptoms.—General symptoms of stomatitis, and, on inspection, a diffuse red swelling of the mucous membrane.

Treatment.—The cause must be removed. Errors of hygiene should be corrected. The diet and the state of the alimentary tract should receive careful attention. The mucous membrane of the mouth should be washed at frequent intervals with cool antiseptic solutions. In mild *catarrhal stomatitis* a solution of boric acid, 5 to 10 grains to the ounce, will suffice. In obstinate cases the mouth, after being carefully cleansed, may be lightly painted with a solution of silver nitrate, 4 grains to the ounce.

APHTHOUS STOMATITIS.

(Follicular Stomatitis; Vesicular Stomatitis.)

Symptoms.—General symptoms of stomatitis, and, on inspection, numerous small white vesicles on the cheeks, lips, and tongue; these vesicles soon break, and leave little shallow ulcers with a red areola.

Prognosis.—Good.

Treatment.—The same as for catarrhal stomatitis. The following application is useful:

℞.	Acidi borici	gr. x–xx
	Glycerini	f℥ss
	Aquæ	q. s. ad f℥ij.—M.

ULCERATIVE STOMATITIS.

This is thought by some to be an infectious disease, because it often occurs in epidemics, and may attack both children and adults when congregated and subjected to bad hygienic conditions.

Symptoms.—General symptoms of stomatitis.

Inspection.—The gums of the lower jaw are chiefly affected. They are swollen, red, and spongy. Linear ulcers, with gray, sloughing bases, soon form, and may extend to the cheek. The glands under the jaw are swollen. In severe cases loosening of the teeth and necrosis of the bone may follow.

Prognosis.—Guardedly favorable.

Treatment.—Hygienic conditions must be improved. Potassium chlorate is almost a specific. It should be used both locally and internally. The dose for a child of three years is from 1 to 3 grains, well diluted, every three hours. The ulcers may be painted with a solution of silver nitrate, 10 grains to the ounce. Tonics, like quinin and iron, are called for in some instances.

PARASITIC STOMATITIS.

(**Thrush; Muguet.**)

Exciting Cause.—Oïdium albicans.

Symptoms.—General symptoms of stomatitis, and, on inspection, numerous milk-white elevations which, on removal, leave a raw surface. The disease may extend to the pharynx, esophagus, and larynx. Microscopic examination reveals the fungus.

Prognosis.—Good.

Treatment.—Everything that comes in contact with the child's mouth should be rendered absolutely clean. Gastro-intestinal derangements should receive attention. The mouth should be cleansed at frequent intervals, especially after feeding, with one of the following solutions: Sodium bicarbonate (1 dram to 5 ounces); sodium hyposulphite (20 grains to 1 ounce); potassium permanganate ($\frac{1}{2}$ grain to 1 ounce).

GANGRENOUS STOMATITIS.

(Cancrum Oris; Noma.)

This form is seen most frequently in debilitated children between the ages of two and six years, and usually follows one of the specific fevers, especially measles and whooping-cough. It may be a sequel to ulcerative stomatitis.

Various micro-organisms have been isolated, especially the diphtheria bacillus and a thread-like parasite of the leptothrix type.

Symptoms.—The general symptoms of stomatitis are marked. The cheek is the part usually affected.[1] Externally, it is swollen, hard, red, and glazed; internally there is noted an irregular, sloughing ulcer. The putrefaction causes an intensely fetid odor. The duration of the disease is from one to three weeks.

Complications.—Perforation, septicemia, lobular pneumonia from aspirated sloughs, and diarrhea from the swallowing of fetid material.

Prognosis.—Grave. In the large majority of cases (85 per cent.) the child dies from exhaustion or complications. Recovery is usually attended with deformity.

Treatment.—The sloughing surface and the tissue immediately surrounding it should be promptly destroyed under anesthesia with the actual cautery or strong nitric acid. After the operation the mouth should be cleansed at frequent intervals with a solution of hydrogen dioxid (1 : 3) or of potassium permanganate (1 per cent.). Concentrated nutritious food, stimulants, and tonics are urgently indicated.

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[1] In girls noma sometimes attacks the vulva.

MERCURIAL STOMATITIS.

(Ptyalism.)

This form of stomatitis is seen in artisans who work in mercury, after the administration of very large doses of mercurials, and after the administration of small doses when there has been an unnatural susceptibility.

Symptoms.—**Premonitory Symptoms.**—Tenderness of the gums, manifested by bringing the teeth forcibly together; redness of the gums near the insertion of the teeth, a metallic taste, and an increase of saliva.

Later Symptoms.—Profuse salivation, fetor of breath, redness, swelling, and tenderness of the gums. The tongue may be similarly affected and protrude from the mouth. In severe cases ulceration of the mucous membrane, loss of teeth, and necrosis of the jaw result.

Treatment.—The administration of mercury should be suspended as soon as the slightest tenderness of the gums manifests itself. The mouth should be frequently rinsed with a saturated solution of potassium chlorate. In severe cases the affected parts may be painted with slightly diluted sulphurous acid or with a saturated solution of iodoform in ether. To check the excessive flow of saliva, atropin ($\frac{1}{120}$ grain) may be given once or twice a day. Morphin may be required at night to relieve pain and to secure sleep. Potassium iodid is recommended to aid in the elimination of the mercury. Tonics may be needed to combat the anemia and exhaustion.

ACUTE TONSILLITIS.

(Amygdalitis.)

Etiology.—Acute tonsillitis occurs at all ages, but it is particularly common in youth.

Exposure to cold and wet usually excites it, and such exposure is very effective when the system is debilitated or the throat is congested from improper use of the voice. Impure air, as the effluvium from foul drains or sewers, apparently may cause it. As a secondary affection it is of frequent occurrence in acute infectious diseases, as scarlet fever, diphtheria, rheumatism, and variola. Streptococci, staphylococci, diphtheria bacilli, or pneumococci may be found in the exudate.

Varieties.—(1) Simple or catarrhal. (2) Follicular or lacunar. (3) Phlegmonous (quinsy).

Symptoms.—The chief symptoms are chilliness, headache and backache, high fever (103°–105° F.), pain in the throat, difficult deglutition, an altered nasal voice, salivation, fetor of the breath, and swelling and tenderness behind the angles of the jaw.

In the *catarrhal* form the tonsils are uniformly swollen, red, and covered with tenacious mucus.

In the *follicular* form the tonsils are red and swollen, and present little yellow spots on their surfaces. These spots correspond to collections of desquamated and degenerated epithelial cells in the lacunæ or crypts of the gland. During convalescence the contents of the lacunæ are often expelled in the form of cheesy pellets having a characteristic unpleasant odor.

In the *phlegmonous* form the tonsils are extremely swollen—often so much that they almost meet; the pain is intense and of a throbbing character. One gland soon becomes larger than the other, softens, fluctuates, and turns yellow from suppuration. Swallowing is almost impossible, the voice is lost, and breathing is difficult.

Complications.—Albuminuria is frequent. Endocarditis, otitis media, and a diffuse erythema occasionally occur. Suffocation from rupture into the larynx and ulceration into the carotid artery are extremely rare terminations.

Diagnosis.—Follicular tonsillitis must be distinguished from scarlet fever and diphtheria.

Scarlet Fever.—The early and persistent vomiting, the very frequent pulse, the "strawberry tongue," and the peculiar punctiform eruption will suggest scarlatina.

Diphtheria.—In this disease there is an ashy-gray membrane, which cannot be readily detached, and which, if removed forcibly, leaves a bleeding surface. The membrane does not remain limited to the tonsils, but soon spreads to the pillars, uvula, and pharynx. In doubtful cases the only criterion is the presence or absence of the Klebs-Löffler bacillus.

Prognosis.—Favorable; accidents are very rare. The duration varies from a few days in the mild catarrhal form to a week or more in the phlegmonous form.

Treatment.—The patient should be confined to a warm room, and if there be much fever, to bed. A mild aperient is indicated at the outset. The diet should be light but sustaining. The sucking of ice affords relief. The most reliable internal remedies are the salicylic compounds and sodium benzoate. These should be given in full doses at frequent intervals.

℞. Ammonii salicylatis . . . . ʒj
Syrupi acaciæ . . . . f℥ss
Aquæ menthæ piperitæ . . . q. s. ad f℥iij.—M.
SIG.—A teaspoonful every three hours for a child of six years.

Guaiac is also recommended. A dram of the ammoniated tincture of guaiac may be given in milk every three hours. Febrile symptoms, if pronounced, may be controlled by small doses of phenacetin or by a combination of aconite and spirit of nitrous ether. The pain may be so intense as to require the use of opium.

Local Treatment.—Externally, cold applications aid in bringing about resolution; if, however, suppuration be inevitable, warm applications should be employed to hasten the process. Antiseptic sprays, like Dobell's solution (see p. 37) or a solution of hydrogen dioxid (1 : 4), are of decided benefit. Direct applications to the surface of the glands of the tincture of ferric chlorid, of a saturated ethereal solution of iodoform, or of dry sodium carbonate are often useful.

℞. Potassii chloratis . . . . gr. xx
Tincturæ ferri chloridi . . . . fʒiij
Glycerini . . . . fʒvj
Aquæ . . . . q. s. ad f℥ij.—M.
SIG.—Use locally.

Scarification, followed by gargling with hot water, is another measure which frequently affords relief.

Pus should be evacuated as soon as its presence can be detected. In the majority of cases it is best to make the incision not in the tonsil itself, but in the soft palate, a little above and to the outer side of the gland.

HYPERTROPHY OF THE TONSILS.

Etiology.—Hypertrophy of the tonsils occurs most frequently in childhood. While it is often excited by repeated attacks of tonsillitis, in some cases there appears to be no other cause than a congenital predisposition.

Pathology.—It may be a true hypertrophy, but in most instances either the glandular structure or the connective tissue predominates; the firmness of the gland increases in proportion to the overgrowth of the latter. The follicles are often dilated and filled with cheesy material which results from the accumulation of fatty degenerated epithelium. Nasopharyngeal catarrh, hyperplasia of the lingual tonsil, and adenoid growths in the nasopharynx are often associated conditions.

Symptoms.—The symptoms consist in mouth-breathing, snoring during sleep, difficult deglutition, a thick voice of a nasal quality, fetor of the breath, impairment of hearing, a listless expression of countenance, mental dulness, and malnutrition. Night-terrors are common. Persistent interference with breathing through the nose gives rise to the following deformities: narrowing of the nostrils, contraction of the superior dental arch, elevation of the hard palate, and, especially, a chest conformation like that of rickets (*pigeon-breast*).

Complications.—Hypertrophy of the tonsils increases the liability to acute catarrh of the nasopharynx, to follicular tonsillitis, and to diphtheria. Chronic catarrh of the middle ear, bronchial asthma, and facial chorea are possible sequels.

Prognosis.—Favorable; if proper treatment be adopted.

Treatment.—Attempts to reduce the enlargement by applying tincture of iodin, tincture of ferric chlorid, alum and glycerin, etc., usually fail. When the glands are very large and the general health is suffering, no time should be lost in resorting to tonsillectomy. Pharyngeal adenoids should also be removed.

Constitutional treatment should not be neglected. It includes systematic bathing, breathing exercises, attention to diet and clothing, and the administration of such drugs as cod-liver oil, hypophosphites, and iodid of iron.

PHARYNGITIS.

ACUTE PHARYNGITIS.

(Acute "Sore Throat"; Simple Angina.)

Definition.—An acute catarrhal inflammation of the mucous membrane of the pharynx, soft palate, and uvula. It is frequently associated with tonsillitis and laryngitis.

Etiology.—Exposure to cold and wet is the most common cause. It may be of rheumatic or gouty origin. It may be excited by local irritants, such as hot drinks or the inhalation of noxious gases.

It is also met with in scarlet fever, measles, and other infectious fevers.

Symptoms.—Chilliness, slight fever with its associated phenomena, stiffness and tenderness of the muscles of the neck, soreness in the throat, painful deglutition, a sensation of dryness or tickling, and a hacking cough. Extension to the larynx may cause hoarseness; to the ear, through the Eustachian tube, deafness. Inspection reveals a red and swollen mucous membrane.

Prognosis.—Favorable.

Treatment.—In mild cases a gargle of potassium chlorate will suffice. In severe cases the application to the throat of cloths wrung out of cold water proves grateful. The sucking of pieces of ice affords much relief. Gargles or sprays of the distillate of hamamelis (50 per cent.) are useful. A spray of menthol, 2 grains to the ounce of liquid petrolatum, is also efficacious. Lozenges containing cocain will often relieve pain and allay the tickling sensation in the throat. The following formula, recommended by Bosworth, answers the purpose admirably:

℞.	Cocainæ hydrochloridi	gr. v
	Extracti krameriæ	gr. ij
	Sodii bicarbonatis	gr. xv
	Extracti glycyrrhizæ	ʒiss.—M.

Fiant trochisci No. xxx.

Internally a mild aperient may be given at the outset. Sodium benzoate (5 grains four times daily) has a beneficial effect. Belladonna with aconite is also recommended. The rheumatic form usually yields promptly to a mild salicylic preparation like salophen (5 to 8 grains three or four times a day).

ANGINA LUDOVICI.

(Ludwig's Angina.)

This is a very grave and rapid form of phlegmonous inflammation of the tissues about the floor of the mouth and sides of the neck. It may occur in the course of various specific fevers, or it may be excited by traumatism or carious processes at the roots of the teeth. It may end in abscess-formation or gangrene, and frequently leads to general septicemia.

CHRONIC PHARYNGITIS.

Etiology.—Chronic "sore throat" may result from repeated acute attacks, from overuse or improper use of the voice, or from the prolonged action of irritants, like tobacco-smoke. It is a frequent attendant upon chronic nasal catarrh and indigestion.

Varieties.—(1) Hypertrophic; (2) atrophic.

Symptoms.—The voice is husky, and its use is followed by distress; secretion is increased, so that there is a constant desire to clear the throat; disagreeable sensations, as fulness, tickling, and the like, are frequently noted.

In the hypertrophic form (granular sore throat, clergyman's sore throat, chronic follicular pharyngitis) the mucous membrane is thick, swollen, traversed by dilated veins, and studded with numerous elevations which correspond to distended follicles and overgrown lymphatic tissue.

In the atrophic form (pharyngitis sicca) the mucous membrane is pale, smooth, glossy, and dry.

Treatment.—The removal of the cause is of prime importance. All sources of local irritation, such as misuse and overuse of the voice, mouth-breathing, excessive smoking, and intemperance in eating and drinking, must be avoided. Patients should be instructed to expel sounds by the aid of the diaphragm and abdominal muscles instead of the muscles of the throat. Nasal obstructions and adenoid growths must be removed. The habit of hawking and scraping to clear the throat should be rigidly interdicted. Digestive disturbances should receive careful attention. Tonics, like iron, strychnin, and cod-liver oil, are sometimes required.

Local Treatment.—The nasopharynx should be kept clean by frequent spraying with an antiseptic alkaline liquid, like Dobell's solution:

℞.	Sodii bicarbonatis	
	Sodii boratis	āā gr. xv
	Acidi carbolici	gr. viij
	Glycerini	fʒij
	Aquæ	f℥viij.—M.

Astringent applications are often of service; one of the following may be employed: Zinc sulphate, 5 grains to the ounce; tannin, 1 dram to the ounce of glycerin; silver nitrate, 10 to 20 grains to the ounce. In the follicular variety it is advisable to destroy the enlarged follicles by means of the galvanocautery, after which the astringent applications may be made.

RETROPHARYNGEAL ABSCESS.

(Retropharyngeal Lymphadenitis.)

This is a suppurative inflammation of the pharyngeal lymphatics, usually secondary to one of the specific fevers, to follicular tonsillitis, suppurative rhinitis, otitis media, or to caries of the cervical vertebræ. It occurs especially in children. It may be recognized by pain in the throat, dysphagia, dyspnea, alteration in the voice, and the detection, on inspection or palpation, of a swelling projecting from the posterior pharyngeal wall.

Treatment.—As soon as pus can be detected it should be evacuated by means of a guarded bistoury, the head of the child being held forward to prevent the escape of the pus into the larynx.

STENOSIS OF THE ESOPHAGUS.

Varieties.—(1) Functional obstruction due to spasm (esophagismus). (2) Organic obstruction.

SPASM OF THE ESOPHAGUS.

(Esophagismus.)

Etiology.—It usually occurs in women as a manifestation of hysteria. It may occur as a symptom of hydrophobia or of chorea. It may be due to reflex irritation originating in the esophagus itself or in some distant organ.

Symptoms of Hysteric Esophagismus.—It is manifested by paroxysmal dysphagia, a sense of constriction in the chest, and sometimes by choking and the regurgitation of food.

Diagnosis.—It may be recognized by the age and sex of the patient, the paroxysmal character of the obstruction, the ease with which a bougie can be passed, the presence of emotional disturbances, the absence of emaciation, and the absence of any other obvious cause.

Prognosis.—Good.

Treatment.—The underlying neurosis should receive appropriate treatment. The systematic passage of a bougie often results in a cure.

ORGANIC ESOPHAGEAL OBSTRUCTION.

Etiology.—(1) An external tumor pressing on the esophagus. This is most commonly an aneurysm. (2) A tumor growing from the esophageal wall, generally a cancer. (3) A cicatrix from ulceration. The ulcer may be due to syphilis or to the ingestion of some corrosive poison, as a strong acid or alkali. (4) A foreign body.

Symptoms.—The chief symptom is slowly increasing difficulty in deglutition, with the regurgitation of food. The esophagus is often much dilated above the constriction, and the food may collect in the pouch thus formed, so that regurgitation may be delayed for several hours. The passage of a bougie meets with a permanent obstruction. There is much loss of flesh.

Diagnosis.—The history of syphilis or of corrosive poisoning will suggest a cicatrix. Aneurysmal obstruction can usually be detected by physical examination. Aneurysm should be excluded before a bougie is passed. The age, cachexia, pain, expectoration of blood-streaked mucus, and involvement of other organs will indicate cancer.

Prognosis.—Depends on the cause. It is unfavorable in aneurysm and cancer. In cicatricial contraction the obstruction may be overcome for an indefinite period.

Treatment.—Aneurysm: Prolonged rest, dry diet, potassium iodid; surgical measures (Moore-Corradi treatment, see p. 196). Cicatricial contraction: Systematic dilatation with graduated bougies. Cancer: In the early stage, the cautious use of a bougie is advisable. In advanced cases the patient may be fed through a tube, and when this is no longer possible, life may be prolonged for a short time by rectal alimentation or by feeding through a gastric fistula.

ACUTE GASTRITIS.

(Acute Gastric Catarrh.)

Etiology.—It may result from—(1) The ingestion of indigestible food, of partially decomposed food, or of excessive quantities of food; (2) from the ingestion of irritant poisons—alcohol, strong acids, or alkalis; (3) it accompanies many of the infectious fevers.

Pathology.—The mucous membrane is red, swollen, and covered with thick mucus. It is sometimes the seat of ecchymosis. The microscopic changes consist in marked mucoid degeneration and cloudy swelling of the epithelial cells, and the infiltration of the interstitial tissues with round cells.

In toxic gastritis there is often extensive sloughing of the gastric mucosa.

Symptoms.—The symptoms vary much in degree. In mild cases there are anorexia, a feeling of discomfort and fulness, eructations, nausea, and, perhaps, vomiting. The tongue is heavily coated. In severe cases the symptoms are more marked, particularly the nausea and vomiting. There may be also moderate fever (102°–103° F.), thirst, herpes, distention of the epigastrium, local tenderness, and considerable prostration. The vomitus is composed at first of sour, fermented food; later, of mucus and bile. Jaundice may follow from the extension of the catarrh to the duodenum and bile-ducts, and diarrhea from its extension to the intestines.

Toxic gastritis is manifested by intense burning pain in the throat, gullet, and stomach, persistent vomiting of food-remnants mixed with blood and mucus, marked abdominal tenderness, and the phenomena of collapse.

Atrophy of the mucosa and cicatricial stenosis of the orifices are common sequels in cases that do not prove immediately fatal.

Diagnosis.—It may resemble the onset of scarlet fever, but the history of contagion, the "strawberry tongue," sore throat, very rapid pulse, and eruption will lead to the recognition of the latter.

Prognosis.—Simple acute gastritis runs a favorable course, and rarely lasts more than a few days.

Treatment.—Absolute rest is essential. If the stomach has not been completely emptied, an emetic, such as warm water or ipecac, should be employed. Locally, a mustard-plaster or a turpentine stupe will aid in relieving distress. As a rule, no food should be given by the mouth until the stomach becomes retentive. Ice, however, may be allowed to quench the thirst. In delicate subjects nutrient enemata will be required. If there is constipation, a mercurial laxative may be given with advantage. Such a combination as the following usually acts favorably:

℞. Hydrargyri chloridi mitis . . . . . . gr. j
Bismuthi subnitratis . . . . gr. xx.—M.
Fiant chartulæ No. vj.
SIG.—One on the tongue every hour, to be followed by a Seidlitz powder, if necessary.

Severe pain, nausea, restlessness, and insomnia are best relieved by opium suppositories. Persistent vomiting may be relieved by bismuth subnitrate (10 grains) combined with creasote ($\frac{1}{2}$ minim), with cocain ($\frac{1}{6}$ grain), or with hydrocyanic acid, as in the following formula:

℞. Bismuthi subnitratis . . . . ʒiij
Acidi hydrocyanici diluti . . . . . . ♏ xxxij
Aquæ . . . . f℥iv.—M.
SIG.—Shake well. A dessertspoonful every three hours.

The following combination of ipecac and nux vomica is often serviceable:

℞. Tincturæ nucis vomicæ
Vini ipecacuanhæ . . . . āā fʒij.—M.
SIG.—Two drops every hour.

After the lapse of twenty-four or thirty-six hours it is generally possible to give bland nourishment by the mouth. Barley-water, champagne with soda-water, milk and lime-water, peptonized milk, and light broths may be given in small quantities at frequent intervals. The return to solid food should always be carried out very gradually.

The treatment of *toxic gastritis* consists in the immediate neutralization of the poison by chemical antidotes, in the evacuation of the stomach (except in the late stages of poisoning by caustics) by the stomach-pump or emetics, and in the administration of demulcents and opium.

9 CHRONIC GASTRITIS.

(Chronic Gastric Catarrh; Catarrhal Dyspepsia.)

Etiology.—It may be excited—(1) By prolonged irritation of the stomach, such as results from errors in diet (excesses in eating and drinking, indigestible food, excessive, irregular meals, deficient mastication, etc.) or from the excessive use of alcohol, tobacco, condiments, or purgatives; (2) by passive congestion the result of chronic heart disease or cirrhosis of the liver; (3) by chronic diseases that disturb metabolism, such as tuberculosis, diabetes, chronic Bright's disease, gout, chlorosis, etc.; (4) by chronic diseases of the stomach itself, such as cancer, ulcer, gastrectasis, etc.

Pathology.—The mucous membrane is of a grayish or slaty color, swollen, and covered with tenacious mucus. The veins are dilated, and there may be ecchymoses.

Microscopically, there is a cellular infiltration in the interstitial tissue. The glands are dilated, elongated, and tortuous, and their epithelium is more or less degenerated and detached. The interglandular proliferation may be so pronounced as to cause great thickening of the mucous membrane (*hypertrophic gastritis*), or, on the other hand, the new-formed fibrous tissue may contract to such a degree as to cause extreme thinning of the coats of the stomach and atrophy or complete destruction of the glandular elements (*atrophic gastritis*).

Symptoms. — The *subjective symptoms* are very variable, and, for the most part, not characteristic. The chief phenomena are furring of the tongue, fetor of the breath, anorexia, fulness and distress, especially at the height of digestion, belching, eructations, heartburn, constipation, headache, vertigo, and attacks of palpitation. Nausea and vomiting are not uncommon. The latter may occur before breakfast or at the height of digestion. If it occurs on rising in the morning, the vomit consists of tough masses of mucus; if it occurs after meals, the vomit is composed of undigested food remnants intimately mixed with more or less glairy mucus. The entire epigastrium may be sensitive to pressure.

The *objective symptoms* are characteristic. Examination of the stomach-contents reveals an excessive secretion of mucus, a marked reduction in the secretion of HCl[1] and of the digestive ferments, and imperfect digestion of albumins. In uncomplicated cases there is no motor insufficiency.

Chronic gastric catarrh rarely terminates in *atrophic gastritis* (*achylia gastrica*), the most important symptoms of which are paroxysmal pain, more or less persistent vomiting, constipation alternating with diarrhea, pronounced emaciation and anemia, and absence of free HCl and of digestive ferments from the stomach-contents after a test-breakfast.

Diagnosis.—Atony of the Stomach.—In simple atony fluids excite as much distress as solids, vomiting is not common, the secretion of mucus is not increased, the secretion of HCl is not usually decreased, and considerable quantities of undigested food can be recovered from the stomach seven hours after a test-meal.

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[1] In rare instances the secretion of HCl is increased.

Hyperchlorhydria.—In this condition the general health is not impaired, the appetite is usually good, there is more or less severe pain shortly after eating, albumins and alkalis relieve the pain, and excess of HCl is found in the stomach-contents; albumin-digestion is good, starch-digestion is retarded, and there is no excess of mucus.

Nervous Dyspepsia.—In this syndrome the severity of the symptoms varies considerably from day to day according to the mental state of the patient, and is not materially influenced by the quantity or the quality of the food; the general health is not often impaired, the nervous symptoms are very prominent, the secretion of the stomach is usually normal, and there is no excess of mucus.

Peptic Ulcer.—The severe, localized paroxysms of pain shortly after eating, the localized tenderness, hematemesis, and hyperacidity will serve to distinguish ulcer from catarrh.

Cancer of the Stomach.—The history, rapid course, cachexia, persistent vomiting, hematemesis, palpable tumor, signs of gastrectasis, and the early absence of free HCl from the gastric juice, with the presence of large quantities of lactic acid and of the Boas-Oppler bacilli, will usually render the diagnosis clear.

Care must be taken to determine whether the catarrh is *primary* or *secondary* to some constitutional or visceral disease.

Prognosis.—The primary forms of chronic gastritis, when not too far advanced, are frequently cured. The prognosis is unfavorable when there is much atrophy of the gastric mucosa. In the secondary forms the prognosis is dependent on that of the primary disease.

Treatment.—The cause should be ascertained and removed if possible. Regularity in the time of meals, slowness in eating, and thorough mastication of food must be insisted upon. The patient should be cautioned against overeating and the taking of large quantities of liquid, especially of iced water, during meals. Overindulgence in alcohol, tobacco, coffee, and tea should be forbidden. The resumption of mental or physical work immediately after meals should also be avoided.

A mixed diet of bland, readily digestible food is required. It may usually include boiled, baked, or grilled beef and mutton, chicken, sweetbread, boiled fish, oysters, soft-boiled or poached eggs, pulled bread, fresh butter, baked potato, young string-beans, small peas, spinach, hearts of celery, thoroughly cooked cereals, calves'-foot jelly, and junket. Tea, coffee, and cocoa may or may not be permissible.

An exclusive milk diet acts exceedingly well in some cases. Systematic lavage is of great value in severe cases, especially when there is excessive secretion of mucus. When lavage cannot be tolerated, the stomach may be cleansed by a glass of hot alkaline water slowly sipped a half-hour or more before breakfast. The following artificial Carlsbad salt may be used as the alkali:

℞. Sodii sulphatis . . . . ʒx
Sodii bicarbonatis . . . . ℥iv
Sodii chloridi . . . . ℥ij.—M.
SIG.—A teaspoonful in a glass of hot water an hour before breakfast.

In mild cases the administration of a bitter—calumba, gentian, nux vomica—some time before meals often proves efficacious. In many cases an alkali may be added with advantage to the bitter, as in the following formula:

℞. Sodii bicarbonatis . . . . ʒiss
Infusi gentianæ compositi . . . . . . f℥vj.—M.
SIG.—A tablespoonful before meals.

When the stomach is highly sensitive, silver nitrate will be found a valuable remedy. It may be given in pill form in combination with hyoscyamus, as in the following formula:

℞. Argenti nitratis . . . . gr. vj
Extracti hyoscyami . . . . gr. x.—M.
Fiant pilulæ No. xx.
SIG.—One pill a half-hour before meals.

Bismuth subnitrate is also of service in such cases. Diluted hydrochloric acid is sometimes serviceable in replacing the natural acid of the gastric juice. In many cases, however, better results are secured from the administration, during meals, of pancreatin with sodium bicarbonate. Flatulence and fermentation may be controlled by such antiseptics as bismuth salicylate, creasote, bismuth-beta-naphthol, etc. The following combination is often of value.

℞.	Creasoti	♏ xx
	Bismuth-beta-naphthol	gr. c
	Pulveris zingiberis	gr. xxx.—M.

Pone in capsulas No. xx.
SIG.—One after meals.

So far as possible, constipation should be overcome by regulation of diet, systematic exercise, and the use of enemas or suppositories.

Change of scene, a sunny climate, good hours, and freedom from business worry and household cares often prove more beneficial than any other measure employed.

ATONY OF THE STOMACH.

(Motor Insufficiency; Myasthenia Gastrica.)

Definition.—Atony of the stomach consists in relaxation of the muscular coat of the stomach and insufficiency of its propulsive powers. It frequently leads to gastrectasis.

Etiology.—Motor insufficiency is of common occurrence. It may be congenital; it may be caused by intemperance in eating and drinking; it may follow acute infections; it may occur in the course of chronic diseases attended by malnutrition; it may appear acutely after traumatism or intense emotional excitement; it may be a complication in other diseases of the stomach, especially in gastroptosis, chronic gastritis, nervous dyspepsia, and hypersecretion.

Symptoms.—In simple atony the chief symptoms are a feeling of fulness and discomfort after meals, especially if the latter have been large, and frequent belching of gas. The severity of the symptoms often bears a definite relation to the quantity of food taken. Fluids are as likely to excite distress as solids. As a rule, there is neither vomiting nor pain. The appetite is usually good, the general health is not seriously affected, and the symptoms entirely abate upon the evacuation of the stomach. There are no signs of gastrectasis. When the intestines are similarly affected, there may be marked nervous symptoms—headache, vertigo, and paresthesia—and considerable disturbance of nutrition.

The diagnosis is rendered certain by the recovery of a considerable quantity of undigested food from the stomach seven hours after the ingestion of the Riegel test-meal.

Prognosis.—Favorable, if the cause can be removed.

Treatment.—The first indication is to remove the cause. The food should be readily digestible, small in bulk, finely divided, and nutritious. Fluids, except in moderate quantities, and coarse vegetables are to be avoided. The diet may include tender meats, eggs, oysters, boiled fish, well-cooked cereals, steamed rice, stale bread, fresh butter, baked potatoes, tender spinach, string-beans, and asparagus-tips. It is rarely necessary to increase the number of meals. Rest for at least an hour after large meals is to be urged. Exercise in the open air and frequent tepid baths are general measures of value. Lavage is unnecessary unless there are gastrectasis and fermentation.

General tonics, especially iron, are often needed. The most useful direct remedies are the bitters (quassia, gentian, and calumba), particularly the tincture of nux vomica, which may be given in doses of from 5 to 10 minims, gradually increased, before meals. Alkalis are indicated when there is hypersecretion. Antifermentatives—bismuth salicylate, beta-naphthol-bismuth, salol, and creasote—are useful in reducing flatulence.

Constipation is best relieved by diet, abdominal massage, and enemas.

NERVOUS DYSPEPSIA.

(Neurasthenia Gastrica.)

Definition.—The characteristic feature of this syndrome is pronounced discomfort during the period of digestion, out of all proportion to the disturbances of gastric secretion or motility. The source of the discomfort appears to be an excessive irritability of the nerves of the stomach.

Etiology.—Nervous dyspepsia usually occurs in those of a distinctly nervous temperament, and mental overexertion, worry, and excesses are potent etiologic factors. It is frequently associated with neurasthenia and hysteria. It may be due to reflex irritation from other organs.

Symptoms.—The tongue is often clean. The appetite is very variable—at one time it is lost, at another it is inordinate, at another it is perverted, the patient craving unnatural food. Pain during the period of digestion is a prominent symptom. It varies in intensity from a feeling of discomfort to the most violent distress. There is rarely tenderness, but the skin over the stomach is often abnormally sensitive.

Belching is common. Vomiting is not frequent. Exaggerated peristaltic movements attended with gurgling sounds (peristaltic unrest) may be perceptible to the patient. Nervous phenomena—headache, vertigo, disturbed sleep, hypochondriasis, lassitude, and palpitation—are conspicuous.

Gastric acidity is usually normal, but there may be subacidity or hyperacidity. In the majority of cases gastric motility is not affected, the viscus emptying itself within the normal period.

The symptoms are usually confined to the period of digestion; they are out of proportion to the disturbance of the digestive functions; they vary greatly from day to day, according to the mood of the patient; and they are not materially influenced by the quality or the quantity of the food.

Prognosis.—Good, when the cause can be removed.

Treatment.—The treatment is largely that of neurasthenia. The avoidance of excitement and of excessive mental work must be enjoined. An extended voyage may effect a cure. In brain-workers systematic exercise in the open air and frequent bathing, followed by friction of the skin, often prove very efficacious. On the other hand, the exhausted and anemic may demand the "rest-cure." The diet should be bland and readily digestible. In many cases milk is an appropriate food. Tonics, like iron and arsenic, are often indicated. Short courses of an unirritating bromid, like that of strontium, sometimes do good. The following combination of antispasmodics is useful in certain cases:

℞.	Zinci valeratis	gr. xxx
	Extracti sumbul	gr. xx
	Arseni trioxidi	gr. $\frac{1}{3}$
	Extracti gentianæ	gr. x.—M.

Fiant pilulæ No. xx.
SIG.—One pill after each meal.

HYPERCHLORHYDRIA.

(Superacidity; Hyperacidity.)

Definition.—These terms are used to designate an abnormal increase in the secretion of hydrochloric acid during the digestive act.

Etiology.—This anomaly of secretion is most frequently seen in neuropathic subjects between the ages of fifteen and forty. Mental overexertion, the excessive use of tobacco, overindulgence in condiments, and insufficient mastication are important predisposing factors. It is a common complication in chlorosis, in gastric ulcer, and in cholelithiasis.

Symptoms.—The symptoms do not appear immediately after eating, but at the acme of digestion, and include sensory irritation, varying in degree from slight discomfort to agonizing pain, with acid eructations, heartburn, thirst, diffuse tenderness over the stomach, and occasionally vomiting. These symptoms are relieved by eating small quantities of albuminous food and by the ingestion of alkalis, and disappear spontaneously upon evacuation of the stomach. They may be continuous or periodic.

The stomach-contents obtained after a test-breakfast are excessively rich in hydrochloric acid, both free and combined. Albumin digestion is rapid. The resting stomach is empty. There is no motor insufficiency.

Prognosis.—In the absence of complications the prognosis is favorable.

Treatment.—The cause should be ascertained and removed, if possible; thorough mastication is imperative. The diet should be unirritating, and composed largely of albuminoid foods. Coarse substances, vinegar, spices, condiments, coffee, and alcohol should be avoided. A moderate amount of water or weak tea at meals is desirable. Fats, in the form of cream and butter, are usually well borne. Starchy foods should be used sparingly and only when thoroughly cooked. It is sometimes desirable to increase the number of meals.

Alkalis, in the form of sodium bicarbonate or magnesia, administered at the height of digestion, relieves the symptoms. Silver nitrate ($\frac{1}{4}$ grain) with extract of belladonna ($\frac{1}{3}$ grain) thrice daily, on an empty stomach, is useful. Silver nitrate (1 : 2000 to 1 : 1000) may also be given as a stomach-douche with advantage. As there is often marked hyperesthesia of the gastric mucosa in these cases, sedatives, like bromids, valerates, and sumbul, are sometimes of service.

GASTROSUCCORRHEA.

(Reichmann's Disease.)

Definition.—This is a functional condition characterized by the secretion of large quantities of gastric juice, even when the stomach is empty. It is often associated with hyperchlorhydria. Two forms have been recognized—(1) the continuous and (2) the intermittent.

Etiology.—The causes of gastrosuccorrhea are the same as those which excite hyperchlorhydria.

Symptoms.—In the continuous form the symptoms appear regularly, but with varying intensity, and consist in more or less severe pain, both at the acme of digestion and in the night; vomiting of large quantities of yellowish, acid fluid, even when the ingesta are no longer in the stomach; marked thirst; acid eructations; and headache, sometimes of a migrainous type. The ingestion of a small quantity of albuminous food usually relieves the pain. Albumin digestion is good, but starch digestion is retarded. The diagnosis is rendered certain by the finding of from 50 c.c. to 500 c.c. or more of gastric juice, *without any admixture of food*, in the stomach before breakfast, particularly if lavage has been practised the night before.

Complications.—Gastrectasis may result from imperfect

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digestion of starches or from spasm of the pylorus excited by excessive acidity. Ulcer may coexist. In rare instances tetany develops.

Diagnosis.—In *hyperchlorhydria* the resting stomach is empty and pain does not occur at night. Care must be taken to exclude *locomotor ataxia*, of which intermittent gastrosuccorrhea may be an early symptom.

Prognosis. — Guardedly favorable in uncomplicated cases. Relapses are common.

Treatment.—This is much the same as that for hyperchlorhydria. The painful attacks may be relieved by the administration of alkalis, or, better, by thorough lavage. Belladonna appears to possess some power to reduce gastric secretion.

GASTRALGIA.

(Gastrodynia; Neuralgia of the Stomach.)

Definition.—Violent paroxysmal gastric pain, occurring independently of any organic disease of the stomach and of any disturbances of secretion or motility.

Etiology.—It is more common in women than in men. Overwork, worry, sexual excesses, abuse of tobacco, reflex irritation, and anemia predispose to it. It may be a symptom of neurasthenia.

Symptoms.—The characteristic features are paroxysms of intense pain, occurring suddenly at irregular intervals, radiating to the chest and back, bearing no definite relation to eating, and lasting from a few minutes to several hours. Vomiting is rare. Pressure over the stomach may relieve the pain, and so may the taking of food.

Diagnosis.—Idiopathic gastralgia must be separated from the paroxysmal pain that occurs in gastric ulcer, gastric cancer, hyperchlorhydria, locomotor ataxia, angina pectoris, and in renal and biliary colic.

Gastric Ulcer.—Pain is excited by food and digestion, disappears upon evacuation of the stomach, is associated with hyperacidity, and often with vomiting, hematemesis, and local tenderness.

Gastric Cancer.—The pain is usually more or less continuous, and is aggravated by digestion. There may be persistent vomiting, hematemesis, cachexia, inacidity with lactic-acid fermentation, and a palpable tumor.

Hyperchlorhydria.—The pain is digestive, and is relieved by alkalis and by albuminous food. Examination of the stomach-contents reveals excess of HCl.

Crises of Tabes.—Unsteadiness of gait and of station, Argyll-Robertson pupil, shooting pains in the limbs, abnormalities of sensation, and abolition of deep reflexes will indicate locomotor ataxia.

Angina Pectoris.—The pain radiates from the heart to the neck and arm, is frequently excited by exertion or indiscretions in diet, is generally of short duration, is often attended with immobility of the body and a feeling of imminent dissolution, and is usually associated with the signs of arteriosclerosis.

Renal Colic.—The pain radiates from the kidney into the ureter of the affected side, and concretions or blood may be found in the urine.

Biliary Colic.—The pain is usually in the right hypochondriac region, and is often accompanied by chill, fever, and jaundice. The liver and gall-bladder may be enlarged and tender.

Prognosis.—Favorable in uncomplicated cases.

Treatment.—*The Attack.*—Hot applications are useful. Galvanization (the anode over the stomach and the cathode near the spinal column) often affords prompt relief. The most generally efficacious remedies are antipyrin (8 grains), brandy (1 to 2 fluidrams), aromatic spirit of ammonia (½ fluidram), chloroform (2 to 5 minims), and diluted hydrocyanic acid (2 minims). These remedies are most efficacious when given in hot water. Such a combination as the following is frequently successful:

℞. Chloroformi . . . . fʒiss
Spiritus ammoniæ aromatici
Spiritus vini gallici
Tincturæ cardamomi compositæ . āā fʒv.—M.
SIG.—A teaspoonful in hot water every fifteen or thirty minutes.

In very severe cases it will be necessary to resort to morphin.

The Interval.—The cause must be ascertained, and, if possible, removed. The habits of the patient must be corrected. Methods of treatment intended to improve the general nutrition are of the greatest value. When there is anemia, iron will be found very useful. Among special remedies arsenic, valerianates, sumbul, quinin, and cannabis indica are available. The following combination often proves efficacious:

℞.	Arseni trioxidi	gr. $\frac{1}{3}$
	Quininæ valeratis	gr. xxx
	Ferri pyrophosphatis	
	Extracti sumbul	aa gr. xx.—M.

Fiant in pilulæ No. xx.
SIG.—One pill after each meal.

In some cases a complete change of scene or enforced rest in bed for a given period is the only means of effecting a cure.

PEPTIC ULCER.

(Round Ulcer of the Stomach; Perforating Ulcer.)

Definition.—A circumscribed loss of tissue in the stomach, usually involving both the mucous membrane and the deeper structures, and characterized clinically by paroxysmal pain, localized tenderness, vomiting, hematemesis, and hyperacidity of the gastric juice.

Similar lesions occur in the duodenum and in the lower end of the esophagus.

Etiology.—It is more common in women than in men. The majority of cases occur between the ages of twenty and forty. Chlorosis and anemia are important predisposing factors.

Duodenal ulcer not infrequently follows large superficial burns.

Pathogenesis.—It is generally admitted that these ulcers are due to the digestive action of highly acid gastric juice upon a part of the stomach that has been devitalized in c uence of embolism or thrombosis with infarction, spasm of the blood-vessels, disease of the vessel-walls, or external injury.

Pathology.—As a rule, the ulcers are single, but they may be multiple. The most frequent seat is the posterior wall, in the lesser curvature, near the pylorus. They have a punched-out appearance, are round or oval in outline, and, if recent, are funnel-shaped, with the apex toward the serous coat. The edges are usually smooth, rarely ragged. They vary in diameter from a few millimeters to several centimeters, and may extend to the muscularis or even to the serosa.

Symptoms.—Symptoms of indigestion are generally present. The characteristic symptoms are:

1. *Pain.*—This is usually paroxysmal, severe, and localized. It may radiate to the back or sides. It is closely associated with eating, reaches its acme at the height of digestion, is aggravated by coarse, very hot, acid and spicy foods, is often affected by certain positions of the body, is arrested by vomiting, and subsides spontaneously upon the natural evacuation of the stomach.

2. *Localized Tenderness.*—Two small areas of tenderness can often be elicited, one in front below the ensiform cartilage, and one behind, in the dorsal region, a little to the left of the spine.

3. *Vomiting.*—This frequently occurs in from one-half to two hours after eating. The vomit usually consists of undigested food and acid fluid.

4. *Hematemesis.*—This occurs in at least one-half of all cases. It proves fatal in about 3 per cent. of the cases of ulcer. The blood is generally fluid and unaltered, but if retained in the stomach for some time, it may have a coffee-ground appearance. Occasionally the blood is discharged entirely by the bowel.

5. *Hyperacidity.*—An increase of HCl is almost invariably noted after a test-meal.

In some cases only the symptoms of dyspepsia are present, while in others all symptoms are absent, the disease passing unrecognized until sudden perforation or profuse hemorrhage occurs.

Sequels.—(1) Perforation. This occurs most frequently in ulcers on the anterior wall and in about 6 per cent. of all cases. (2) General or circumscribed peritonitis. General peritonitis is usually the result of perforation; circumscribed productive peritonitis is a conservative process and results from the direct extension of the inflammatory process through the stomach-walls. (3) Subphrenic abscess. This is usually the result of perforation after the formation of adhesions. (4) Stenosis of the pylorus, stenosis of the cardia, or hour-glass constriction of the stomach may result from the contraction of cicatrices. (5) Cancer not infrequently develops on the basis of an old ulcer.

Diagnosis.—*Hyperchlorhydria.*—In this condition the pain does not occur so regularly nor so soon after eating; it is not modified by position, but is often completely relieved by eating albuminous food. Hematemesis is absent and there are no tender spots.

Gastralgia.—In this affection the pain occurs at irregular intervals, is not dependent upon eating (often occurring when the stomach is empty), is relieved by pressure, and is not associated with tender spots, hematemesis, or hyperacidity.

Cancer of the Stomach.—The history, rapid course, advanced cachexia, palpable tumor, vomiting of large quantities of undigested food at long and irregular intervals, coffee-ground vomit, abundance of lactic acid with Boas-Oppler bacilli, and the absence of free hydrochloric acid will point to cancer.

Duodenal Ulcer.—In this disease the pain is further to the right and occurs later after the meals, the blood is usually evacuated through the bowel, and there is no vomiting.

Cholelithiasis.—In this condition the pains appear more suddenly, occur at more irregular intervals, and often independently of eating, usually radiate toward the right shoulder, and are often associated with swelling and tenderness of the liver, enlargement of the gall-bladder, and slight jaundice.

Prognosis.—Guardedly favorable in recent cases. The mortality in all cases is from 8 to 10 per cent. Some ulcers run a rapid course and end fatally in hemorrhage or perforation; others, even without treatment, persist for many years. Relapses are not uncommon.

Treatment.—Rest and appropriate diet are the most important factors in the treatment. The rest should be kept up for from six to twelve weeks, and for the first two or three weeks of this period the patient should be confined to bed. If hemorrhage has recently occurred or if vomiting be urgent, it is advisable to withhold all food from the stomach for a few days and to nourish the patient by means of nutritive enemas.

After the pain and vomiting have sensibly abated, feeding by the mouth should be resumed. The diet should consist of milk, buttermilk, beef-juice, animal broths, egg-white, and thin pap. As soon as the gastric symptoms have completely disappeared, which will rarely be before the lapse of three or four weeks, the patient may be allowed such articles as soft-boiled eggs, scraped beef, boiled sweetbreads, the tender part of oysters, white meat of chicken, well-made gruel, and custard pudding.

The most useful drugs are alkalis, silver nitrate, and bismuth subnitrate. The alkalis are useful in overcoming the superacidity of the gastric juice. Sodium bicarbonate is one of the best; it may be combined with magnesia or chalk, according as there is constipation or diarrhea. Artificial Carlsbad salt (see p. 44) is an excellent alkaline laxative; of this, a teaspoonful or more may be given in a half pint of hot water in the early morning. Silver nitrate and bismuth subnitrate are valuable remedies and may be used alternately, each for a period of a week or ten days. They may be prescribed as follows:

℞. Argenti nitratis
Extracti belladonnæ . . . . āā gr. vj.—M.
Fiant pilulæ No. xx.
Sig.—One pill half an hour before meals.

℞. Bismuthi subnitratis . . . . ʒvj
Acidi hydrocyanici diluti . . . . . . ♏xxiv
Aquæ . . . . f℥vj.—M.
Sig.—A tablespoonful three or four times, half an hour before meals.

Pain and vomiting usually yield to complete rest, rectal feeding, and the administration of silver nitrate or bismuth subnitrate. In some cases it may be necessary to use morphin hypodermically. Externally, stupes or sinapisms are sometimes useful. The treatment of hematemesis is considered on page 63.

Surgical Treatment.—In all cases of perforation an operation should be done at the earliest possible moment. When life is threatened by repeated hemorrhage, operation in the interval between the attacks offers the best method of relief. Again, an operation (gastro-enterostomy, pyloroplasty, or partial gastrectomy) should be considered if the disease does not yield to medical treatment and the life of the patient is endangered by malnutrition.

CANCER OF THE STOMACH.

Etiology.—**Sex.**—Cancer of the stomach is somewhat more common in men than in women.

Age.—The majority of cases occur between the ages of forty and sixty. It is rare before thirty.

Heredity.—About 8 per cent. of the cases appear to be hereditary.

Prolonged Irritation.—Cancer sometimes develops on the basis of an old ulcer.

Pathology.—Cancer of the stomach is almost always primary. The pylorus is the part most frequently attacked. After the pylorus the points of predilection are the lesser curvature and cardia. The following varieties are encountered: Scirrhus or hard cancer, medullary or soft cancer, adenocarcinoma (cylindric-celled epithelioma), colloid cancer, and squamous-celled epithelioma. Ulceration is rare in scirrhus, but common in medullary cancer and adenocarcinoma. Colloid cancer appears most commonly as a diffuse infiltration of the stomach-wall. Squamous-celled cancer is rare, and occurs only at the cardia.

Owing to stenosis of the pylorus the stomach is usually dilated. Stagnation of the stomach-contents and the absence of hydrochloric-acid secretion favor the development of lactic-acid fermentation.

Symptoms.—Symptoms of dyspepsia are generally present. The characteristic phenomena are:

1. *Pain.*—This is rarely intense; though aggravated by eating, it is often more or less continuous. It may radiate to the back.

2. *Vomiting.*—This is very common. When the pylorus is obstructed, the vomiting is persistent and occurs long after eating, sometimes at intervals of several days. The vomit is frequently large in amount, and is composed chiefly of undigested food and turbid fluid. It very rarely contains sarcinæ, but long, thread-like bacilli (*Boas-Oppler bacilli*) are almost constantly present and possess some diagnostic significance.

3. *Hematemesis.*—As the bleeding is slight and the blood remains for some time in the stomach, the vomit in many cases acquires a coffee-ground appearance.

4. *Cachexia.*—The anemia, weakness, and emaciation are often disproportionate to the loss of nourishment.

5. *Palpable Tumor.*—A movable, tender mass can be detected sooner or later in a large proportion of all cases.

6. *Absence of HCl with Lactic-acid Fermentation.*—The absence of free HCl and the presence of large quantities of lactic acid, while not peculiar to cancer, are strongly indicative of the disease.

In addition to these features the symptoms and signs of *gastrectasis* are frequently present.

Complications and Sequels.—Metastases in neighboring structures—liver, lymph-glands, pancreas, and peritoneum—are of common occurrence. Ascites and edema are occasionally encountered. Perforation, subphrenic abscess, tetany, venous thrombosis, multiple neuritis, and coma (from oxybutyric-acid intoxication) are rare complications.

Diagnosis.—The differential points between cancer and ulcer and cancer and chronic gastritis have already been considered.

Prognosis.—The disease is almost invariably fatal. The average duration of life is from one to two years. Marked temporary improvement frequently occurs under treatment and may prove very misleading.

Treatment.—In the early stages of the disease, when the pylorus is still patulous, a mixed diet of readily digested food is often well borne. Later, when there is retention, food should be selected that will make small demands on the stomach and that will leave little residue. Bitters—calumba, gentian, condurango—are sometimes employed with advantage. In many cases, but by no means invariably, hydrochloric acid and pepsin are useful. Lavage affords the best means of relieving the distressing symptoms resulting from retention. Vomiting not dependent upon retention may be treated with such remedies as carbonated water, hydrocyanic acid, creasote, cerium oxalate, and bismuth subnitrate. In obstinate cases rectal feeding may be required for a time. Acid eructations and flatulency are sometimes relieved by antacids and internal antiseptics, but generally lavage is much more effective. Pain will require opium, sedatives like hydrocyanic acid or chloroform, and hot applications.

Early operative interference may prolong life for several months or several years.

DILATATION OF THE STOMACH.

(Gastrectasis.)

Etiology.—Gastrectasis may result from—(1) Atony of the stomach-walls (see p. 45); (2) from stenosis of the pylorus.

Stenosis of the pylorus may be caused by—(*a*) Congenital stricture; (*b*) carcinoma of the pylorus; (*c*) cicatrix from ulcer; (*d*) hypertrophy of the pylorus from gastric catarrh or frequent spasm excited by hypersecretion; (*e*) pressure from without, as by tumors, adhesions, floating kidney, etc.

Pathology.—All degrees of dilatation are encountered. The most severe forms are usually the result of pyloric stenosis. In atonic dilatation the stomach-walls are thin and atrophic; in dilatation from obstruction there may be marked muscular hypertrophy at the pyloric end.

Symptoms.—These vary with the cause and the degree of dilatation. In well-marked cases the chief symptoms are a feeling of fulness and discomfort after meals, frequent belching and acid eructations, increased thirst, constipation, deficient urination, and more or less emaciation. Owing to reflex irritation or autointoxication nervous symptoms often develop.

Vomiting is a characteristic symptom, especially when there is stenosis of the pylorus. It occurs long after meals, sometimes at intervals of several days. The vomit is often excessive in amount, is sour and fermented, and on standing separates into a sediment of undigested food and a supernatant liquid, which is turbid and frothy. Not infrequently the vomit contains remnants of food that was eaten several days before. Microscopic examination may reveal, in atonic dilatation, numerous yeast-cells and sarcinæ, and in cancerous dilatation, the thread-like bacilli of Oppler.

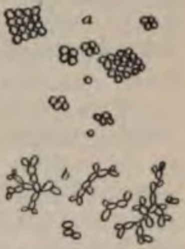

FIG. 1.—*a*, Sarcinæ ventriculi. *b*, Torulæ cerevisiæ.

Physical Signs.—*Inspection.*—The abdomen may be unduly prominent. In some cases the outlines of the enlarged stomach are distinctly visible. Peristaltic waves extending from left to right are frequently seen, especially in stenotic dilatation.

Palpation.—When the stomach-walls are sufficiently tense, the boundaries of the organ may be determined by palpation. In many cases of obstructive dilatation a tumor can be felt at the pylorus.

Percussion.—When the stomach contains fluid, an area of dulness is found at the level of the umbilicus, or below it when the patient is erect, but not when he is lying down. This area of dulness also disappears upon the complete evacuation of the stomach.

After artificial inflation of the stomach with air or carbonic-acid gas percussion reveals an increased area of gastric tympany.

Auscultation.—The detection of splashing sounds over the stomach in the morning before breakfast or seven hours after a Riegel test-meal points to dilatation.

Mensuration.—Normally a rigid sound can be inserted a distance of 24 inches (60 cm.) from the incisor teeth; in dilatation it may be inserted as much as 28 inches (70 cm.). As the depth to which the sound can be passed is frequently increased also in gastroptosis, this sign is unreliable.

X-rays.—Skiagraphs taken after the ingestion of large quantities of bismuth subnitrate suspended in mucilage of acacia have proved of value in the diagnosis of gastrectasis.

Examination of the Stomach-contents.—The detection of remnants of food in the stomach in the morning after a simple supper renders the diagnosis of severe motor insufficiency certain, and that of dilatation highly probable.

Complications.—Gastroptosis is a common complication; it may be secondary to the dilatation or the cause of it. Tetany is a rare, but serious, complication.

Diagnosis.—*Gastroptosis.*—In uncomplicated gastroptosis the position of the stomach is lower down than normal and more or less vertical, but the area of gastric tympany is not increased nor are there present any signs of motor insufficiency.

Type of Dilatation.—A rapid onset, persistent vomiting, pain, a high degree of motor insufficiency with marked dilatation, a tardy inflow and a rapid outflow of water during lavage, active gastric peristalsis, a palpable tumor at the pylorus, and rapid emaciation suggest pyloric obstruction.

Prognosis.—This depends upon the cause, degree, and duration of the dilatation. In pyloric obstruction the prognosis is very grave, although operation may lead to cure, especially in benign forms. Even in atonic dilatation the prognosis must be guarded if the process is advanced and there is pronounced motor insufficiency.

Treatment.—The food should be nutritious, small in bulk, and readily digestible, and, in advanced cases, should be given in small amounts at frequent intervals. Liquids should never be given in large quantities. In severe grades of dilatation, to prevent the tissues from losing water, it is advisable to introduce fluids in the form of water and meat-broth by the bowel. To prevent retention, to control fermentation, and to cleanse the stomach, no measure is so useful as methodic lavage. When there is considerable retention, the lavage should be performed daily, preferably in the early morning. A carefully adjusted abdominal bandage nearly always affords comfort and gives mechanical support to the stomach. In cases due to atony exercise in the open air, hydrotherapy, and, unless there be marked gaseous fermentation, abdominal massage are valuable aids. Faradization of the stomach may also be used to promote muscular contraction. In dilatation from muscular relaxation nux vomica is very useful. Such remedies as creasote, salol, and bismuth-beta-naphthol are sometimes of service in checking fermentation, but the relief they afford is not to be compared to that obtained by systematic lavage. Constipation is best treated by simple enemas or by glycerin suppositories.

Surgical Treatment.—In the large majority of cases of non-obstructive dilatation medical treatment suffices. Occasionally, however, surgical intervention is demanded on account of persistent suffering and progressive emaciation. The operation indicated in these cases is gastroplication. In cases of pyloric obstruction of a benign character an operation is indicated when it is impossible to maintain nutrition by proper medical treatment. As Loreta's digital divulsion of the pylorus has been largely abandoned, owing to its high mortality (31.1 per cent.), there may be said to be but two operations available—pyloroplasty and gastro-enterostomy. The treatment of pyloric cancer is considered on page 58.

GASTROPTOSIS AND ENTEROPTOSIS.

(Glénard's Disease.)

Definition.—Prolapse of the stomach and transverse colon caused by congenital or acquired weakness of the abdominal muscles and ligaments.

Etiology.—The condition is much more common in women than in men. Tight lacing, repeated pregnancies, abnormal formation of the thorax, enlargement of other abdominal organs, gastrectasis, and constitutional weakness are important predisposing factors.

Symptoms.—The chief objective feature is a more or less pronounced downward displacement of the pylorus consequence of which the stomach assumes a vertical or subvertical position. Dislocation of the whole stomach downward is rare. Dilatation of the pyloric extremity is a common sequel. The position and size of the viscus can be determined accurately only after artificial inflation. The transverse colon shares in the downward displacement of the stomach and occupies a position immediately below the greater curvature. Ptosis of other abdominal organs, especially of the right kidney and liver, is also present in many cases. Separation of the recti muscles is often seen. A floating tenth rib is less frequent.

The subjective symptoms are those of motor insufficiency or atony of the stomach,—fulness and distress after meals, splashing, gaseous eructation, pain in the back, and constipation,—combined, in many cases, with more or less marked neurasthenia.

Treatment.—The diet should be adapted to the digestive and motor powers of the stomach. Mechanical support of the pylorus by means of a snugly fitting abdominal bandage affords relief in mild cases. In severe cases, especially when the nervous phenomena are pronounced, a modified rest-cure may prove effective. Lavage is not indicated unless there is general dilatation with retention or excessive secretion of mucus. In very obstinate cases surgical intervention should be considered.

HEMATEMESIS.

(Gastrorrhagia.)

Etiology.—Hemorrhage from the stomach may result from—(1) Traumatism; (2) gastric ulcer; (3) gastric cancer; (4) venous engorgement of the stomach following cirrhosis of the liver, primary splenomegaly, or chronic heart disease; (5) acute gastritis; (6) blood dyscrasia, as in scurvy, purpura, severe infections, and grave anemias; (7) rupture of an aneurysm; (8) swallowing of blood from the nose, mouth, or throat; (9) vicarious menstruation; (10) hysteria.

Symptoms.—The quantity of blood that is vomited varies considerably: rarely a quart or more is lost. In many cases a portion of the blood escapes through the bowel. The blood is usually dark, is often mixed with food, has an acid reaction, and may be fluid or clotted. If the hemorrhage is severe, the symptoms of acute anemia develop—pallor, weakness, vertigo, tinnitus aurium, dimness of sight, syncope, and convulsions.

Diagnosis.—*Hemoptysis.*—The blood is coughed up; it is usually bright red, frothy, and alkaline in reaction; subsequent expectorations are tinged with blood, and the associated symptoms and signs point to pulmonary or cardiac disease.

Prognosis.—Hematemesis is rarely so severe as to cause death. The most dangerous hemorrhages are those that occur in cirrhosis of the liver, splenomegaly, and aneurysm.

Treatment.—In the treatment of hematemesis absolute rest is essential. No food of any kind should be given by the mouth. An ice-bag should be applied over the stomach, and morphin should be given hypodermically. The application of firm bandages to the four extremities may act favorably. Ergot and such drugs as tannic acid, iron sulphate, and lead acetate are of very doubtful utility. A solution of adrenalin chlorid (1 : 1000) is worthy of trial. Twenty minims may be given in half an ounce of water every hour. When the bleeding is prolonged, three or four ounces of a solution of gelatin (10 to 15 per cent.) may be given several times a day.

Collapse following hemorrhage will call for diffusible stimulants, the external application of heat, and the subcutaneous or intravenous injection of warm saline solution.

HABITUAL CONSTIPATION.

Definition.—Infrequent or difficult evacuation of the feces.

Etiology.—The chief causes are: (1) Many general diseases that lessen the intestinal secretions or inhibit peristalsis, such as the acute fevers, anemia, diabetes, neurasthenia, hysteria, and organic affections of the brain and spinal cord. (2) Many diseases of the digestive tract—chronic gastritis, gastrectasis, gastric cancer, obstructive jaundice, and chronic intestinal catarrh. (3) Sedentary habits. (4) Improper food. (5) Atony of the bowel and weakness of the abdominal muscles. (6) Muscular spasm excited by irritable prostate, uterine disease, ulceration of the rectum, or stricture.

Symptoms.—Some persons continue to enjoy excellent health even though their bowels are evacuated at very infrequent intervals. Generally, however, retention of fecal matter in the intestines longer than is customary with the individual gives rise to unpleasant symptoms, common among which are headache, dizziness, mental sluggishness, lassitude, fetor of the breath, a coated tongue, and anorexia.

Sequels.—Severe persistent constipation may lead to piles, fissure, ulceration of the colon, diarrhea from irritation, or fecal impaction.

Treatment.—The removal of the cause is a matter of the first importance. Cathartics should be avoided, if possible. In some cases the activity of the bowels is restored by repeated daily attempts at defecation at some special hour. Systematic exercise and cold bathing are of the greatest benefit. Abdominal massage, especially digital kneading in the direction of the colon, is often quite effectual.

Unless the state of digestion offers a contraindication, such laxative articles of food as green vegetables, oatmeal, cornmeal, whole-wheat bread, oils, and cooked fruits should be ordered. Water-drinking should be encouraged. In mild cases a glass of cold water before breakfast may suffice.

General tonics, like iron and strychnin, are sometimes needed. Mineral waters, like Friedrichshall, Hunyadi János, or the milder Saratoga or Bedford waters, are very useful, but possess no special advantages over the saline laxatives (sodium phosphate or Rochelle salt), when the latter are taken in small amounts well diluted. Enemas of soapy water or of glycerin or suppositories of gluten, soap, or glycerin, often prove highly satisfactory. Vegetable cathartics are usually necessary in obstinate cases. The mild ones should always be tried first, and even with these considerable care should be exercised lest the patient comes to rely upon drugs to the exclusion of the hygienic and dietetic measures already indicated. Of the mild laxatives, cascara sagrada is one of the best: from 10 to 30 minims of the fluid extract, or a corresponding dose of an agreeable elixir, may be administered at bedtime and repeated, if necessary, in the morning.

In many cases a combination of several laxatives (rhubarb, aloes, podophyllum, euonymin, and colocynth) acts better than any one singly. As adjuvants, nux vomica or physostigma may be added to overcome intestinal atony, and belladonna or hyoscyamus to prevent griping. The most suitable combination must be determined in each case by experience. A pill, like one of the following, will generally prove satisfactory:

℞. Aloini . . . . gr. iv
Strychninæ sulphatis . . . . gr. $\frac{1}{3}$
Extracti belladonnæ . . . . gr. iij.—M.
Fiant pilulæ No. xxiv.
SIG.—One pill at bedtime.

℞. Pulveris rhei
Extracti rhamni purshianæ . . . āā gr. xxiv
Extracti euonymi . . . . gr. xij
Extracti physostigmatis
Extracti belladonnæ . . . . āā gr. iv.—M.
Fiant pilulæ No. xxiv.
SIG.—One pill at bedtime.

INTESTINAL COLIC.

(**Enteralgia; Tormina.**)

Definition.—Intestinal pain of a spasmodic character.

Etiology.—It usually results from irritating food, flatulence, or fecal accumulation. It is sometimes of a rheumatic or gouty origin. It is a common symptom of structural lesions of the bowel—enteritis, dysentery, appendicitis, intestinal obstruction. It is an important symptom in chronic lead-poisoning. It may be reflex from disease of the ovaries, uterus, liver, vertebræ, etc. It may occur as a crisis of locomotor ataxia.

Symptoms.—Paroxysms of severe pain of a twisting character, centering around the umbilicus, and relieved by pressure. The abdomen is usually distended. Severe attacks may lead to collapse, indicated by cold sweats, pinched features, feeble pulse, and vomiting. The attack lasts from a few minutes to several hours, and usually ends with a discharge of flatus.

Diagnosis.—**Lead Colic.**—History, blue line on the gums, retracted abdominal walls, wrist-drop, and lead in the urine.

Biliary Colic.—Pain radiating from the liver to the back, jaundice, local tenderness, and calculus in the stool.

Renal Colic.—Pain extending from the kidney along the ureter to the penis and testicle, frequent micturition, blood or calculus in the urine.

Rheumatism of the Abdominal Muscles.—Pain is superficial, persistent, and increased by pressure and movements of the body.

Chronic Appendicitis.—Localized tenderness (McBurney's point), muscular rigidity, and induration.

Intestinal Obstruction. — Localized tenderness, more or less continuous pain, persistent constipation, and incessant vomiting, often stercoraceous.

Treatment.—The indications are to relieve pain and to remove the cause. Turpentine stupes are useful. In severe cases it will be necessary to give morphin ($\frac{1}{8}$ to $\frac{1}{4}$ grain) and atropin ($\frac{1}{100}$ grain) hypodermically. Carminatives—peppermint, ginger, oil of cloves, Hoffman's anodyne—often afford relief.

Colic excited by irritating food or fecal accumulation is promptly relieved by saline or mercurial purges.

DIARRHEA.

Definition.—A condition in which the stools are too frequent or too liquid. Like dyspepsia, it is a symptom of many pathologic conditions.

Etiology.—(1) It results from inflammation of the intestines—enteritis, ileocolitis, dysentery (inflammatory diarrhea). (2) It is a symptom of certain infectious diseases, such as typhoid fever and cholera (symptomatic diarrhea). (3) It may be excited by cathartic drugs. (4) It often occurs as a final symptom in cachectic states, as in cancer, diabetes, and chronic Bright's disease (colliquative diarrhea). (5) It sometimes marks the crisis of acute infections, such as typhus fever and pneumonia (critical diarrhea). (6) It may result from nervous excitement (nervous diarrhea).

INTESTINAL CATARRH.

(Diarrhea; Catarrhal Enteritis.)

Etiology.—Warm weather, childhood, improper food, and bad hygienic surroundings are general predisposing factors. The disease is usually excited by irritating products in the intestinal canal or by sudden changes in temperature. Poisons produced in the decomposition of milk and other foods by bacteria are the most common excitants. Inorganic poisons (arsenic, antimony, mercury) may also induce acute diarrhea.

Pathology.—The mucosa is swollen, slightly injected, and covered with a mucous exudate composed of desquamated and degenerated epithelial cells and leukocytes. The lymph-follicles are enlarged and occasionally ulcerated.

Chronic enteritis may result from acute attacks or from passive congestion in consequence of heart or liver disease. The mucous membrane is pigmented, and, in the early stages, usually thickened from proliferation of the fixed connective-tissue cells. In the later stages atrophy may ensue from destruction of the glands and shrinking of the stroma.

Symptoms.—**Acute Enteritis.**—The chief symptoms are slight fever, with its attending phenomena, colicky pains, rumbling noises (borborygmi), and frequent thin stools, of a yellowish or greenish color, offensive, and containing undigested food. The number of stools varies from three to twelve or more a day. The attack usually lasts from a few days to a week.

Chronic enteritis is manifested by frequent liquid stools, which vary in color and character according to the seat of the catarrh, colicky pains, impaired nutrition, and anemia. The presence in the stools of much undigested food (lientery) indicates involvement of the small bowel, and the presence of much mucus, involvement of the large bowel.

Membranous Enteritis.—This term has been applied to two conditions: (1) A true croupous enteritis, which is associated with the formation of a false membrane, and which is seen in cachectic states, in acute infectious diseases, and as a result of mineral poisoning. (2) Mucous colic, or mucous colitis, a chronic form of colitis usually occurring in women of a marked nervous temperament, and characterized by paroxysms of severe pain and the discharge of gray, translucent casts which, however, are not membranous, but mucoid in character.

Diagnosis.—**Dysentery.**—The small mucous and blood discharges and the severe tenesmus will indicate dysentery.

Ileocolitis.—This disease may be separated from simple enteritis by the continued high fever, more frequent discharges, the presence of blood and mucus, the tenesmus, and the greater prostration.

Peritonitis.—This disease is readily distinguished from enteritis by the more intense pain and tenderness, the greater tympany, the marked constitutional disturbance, the constipation, and the immobility of the patient.

Typhoid Fever.—The gradual onset, nose-bleed, splenic enlargement, characteristic fever, Widal reaction, and eruption will lead to the recognition of typhoid fever.

Prognosis.—Favorable in uncomplicated cases. Chronic diarrhea often persists for many years and is very resistant to treatment.

Treatment.—*Acute Diarrhea in Adults.*—Rest in bed and the substitution of bland nourishment for the ordinary diet are all that is required in many cases. Boiled milk, milk and arrow-root, and mutton, veal, or chicken broth are suitable foods. If the patient is seen at the outset and there is reason to believe that irritant material is still present in the bowel, it is advisable to administer an unirritating purgative, such as castor oil, Epsom salts, or fractional doses of calomel. Occasionally a second dose of the purgative may be given with benefit. Externally, stupes or sinapisms are frequently efficacious. If the diarrhea continues, opium and mild astringents, like bismuth subnitrate and chalk, are indicated. They may be combined advantageously with antiseptics, as in the following formulas:

℞. Morphinæ sulphatis . . . . gr. j
Salol . . . . gr. xxx
Bismuthi subnitratis . . . . ℥ss.—M.
Fiant chartulæ No. xij.
SIG.—One powder every three hours.

℞. Bismuthi subsalicylatis . . . . ʒiss
Cretæ præparatæ . . . . ʒiiss
Tincturæ opii camphoratæ . . . . . . f℥j
Pulveris acaciæ . . . . q. s.
Aquæ cinnamomi . . . . . . q. s. ad f℥vj.—M.
SIG.—A tablespoonful every three hours.

Chronic Diarrhea.—The cause must be ascertained and removed, if possible. The diet, clothing, habits, occupation, and mode of living of the patient should receive careful attention. No definite rules can be laid down in reference to the diet. When the disease is not very severe and is confined for the most part to the colon, a selected mixed diet may be allowed. Many patients do well upon an exclusive milk diet. Foods that are bulky and leave much residue are always inadmissible.

Protection of the body against chilling is of vital importance. Woolens should be worn next to the skin. A snugly fitting abdominal bandage may be worn as an additional safeguard. Rest in bed is sometimes essential. When the general nutrition is not too much impaired, a change of air and scene may prove very beneficial.

Mineral astringents, especially bismuth subnitrate (30 to 40 grains), silver nitrate ($\frac{1}{4}$ to $\frac{1}{2}$ grain), copper sulphate ($\frac{1}{4}$ to 1 grain), and lead acetate (1 to 3 grains) are of service.

Intestinal antiseptics—salol, bismuth salicylate, betanaphthol-bismuth—are useful adjuvants. Opium is often required in acute exacerbations. When the disease is situated chiefly in the colon, irrigation of the bowel two or three times a week with a solution of silver nitrate (10 to 20 grains to 1 pint) is especially to be recommended.

Acute Diarrhea in Infants.—The first indication is to withdraw the milk at once, and to withhold it for several days or until the stools become quite natural. Indeed, in many cases it is well to suspend all nourishment for the first twenty-four hours, allowing nothing by the mouth but barley-water or plain boiled water. Subsequently, albumin-water, fresh beef-juice, veal broth, or a liquid peptone preparation may be given in lieu of milk. Milk feeding should always be resumed very gradually. Absolute rest in the recumbent position is essential. Removal to the seashore or mountains is often of the greatest benefit.

To remove irritant matter from the bowel, castor oil or calomel should be given, preferably the latter when the stomach is sensitive. In most cases it is necessary to follow the purge with a sedative astringent like bismuth subnitrate or chalk. From 5 to 10 grains of one of these drugs may be given every two or three hours with an intestinal antiseptic (salol, beta-naphthol-bismuth, bismuth salicylate); some such combination as the following may be ordered:

℞.	Bismuthi subnitratis	ʒ ij–iv
	Salol	gr. xxiv
	Misturæ cretæ	f℥ iij.—M.

SIG.—A teaspoonful every two hours.

A more active astringent, like tannalbin or tannigen (2 to 3 grains), may be given in addition to the bismuth subnitrate or chalk when the discharges are exceedingly profuse and watery.

Opium is often of great value, but extreme caution must be exercised in its use. It is called for when the diarrhea continues in spite of the thorough unloading of the bowel and the administration of mild astringents. From 3 to 5 minims of paregoric may be given every two, three, or four hours, according to circumstances. When the stomach is unretentive, laudanum (1 to 2 minims) may be given by enema.

ACUTE ILEOCOLITIS.

Definition.—An acute inflammatory disease involving the ileum and colon and affecting especially the lymph-follicles.

Etiology.—It occurs most frequently in artificially fed children. Warm weather and bad hygienic surroundings are important predisposing factors. It sometimes follows acute infections, like measles.

Micro-organisms undoubtedly play an important rôle in the process. In a number of instances the Bacillus dysenteriæ of Shiga has been recognized.

Pathology.—The mucous membrane is swollen and edematous. The solitary and agminated glands are much enlarged and often ulcerated. In some cases the colon is covered with a pseudomembrane.

Symptoms.—The chief features are pronounced fever (102° to 104° F.), abdominal distention and tenderness, tenesmus, frequent small stools of a greenish-yellow color and mixed with mucus and blood, and sometimes with membranous shreds. There is rapid loss of weight and strength. Death is frequently preceded by delirium, stupor, convulsions, and coma (spurious hydrocephalus).

Diagnosis.—The differential diagnosis between ileocolitis and *catarrhal enteritis* has already been considered.

Cholera infantum may be recognized by the rapid onset, constant vomiting, profuse serous stools, great thirst, high rectal temperature, and rapidly developing collapse.

Prognosis.—Grave; mild cases recover in from two to three weeks. Strength is regained slowly and relapses are common.

Treatment.—This is much the same as that of acute catarrhal enteritis. A change of air is very desirable. Stimulants are often required. From 10 to 20 minims of whisky may be given every two, three, or four hours. Intestinal irrigation is an important part of the treatment. Twice a day the colon should be thoroughly flushed with sterile water containing a dram of benzoate of sodium to the pint. After the irrigation an enema of thin mucilage (2 fluidounces) and bismuth subnitrate (2 drams) may be given every three or four hours. Extreme tenesmus is sometimes relieved by small suppositories containing ¼ of a grain of cocain (Rotch).

CHOLERA INFANTUM.

Definition.—An acute gastro-intestinal affection characterized by severe choleriform symptoms. Compared with acute catarrhal enteritis and ileocolitis, it is a rare disease.

Etiology.—Hot weather, faulty feeding, bad hygienic surroundings, dentition, and indigestion are important predisposing factors. The disease is probably excited by a specific micro-organism.

Pathology.—Beyond a slight catarrh of the gastrointestinal tract there are no gross lesions. The grave constitutional symptoms are no doubt due to the absorption of a bacterial poison.

Symptoms.—The symptoms develop rapidly. Vomiting and purging begin almost simultaneously and become incessant. The stools are thin and watery and have a musty odor and an alkaline reaction. Thirst is intense; there is great restlessness; the pulse is rapid and feeble; the surface temperature is low, but the rectal temperature is very high (105° to 106° F.); the urine is almost suppressed. Collapse soon follows, and is indicated by pinched features, hollow eyes, sunken fontanel, pallid skin, and cold surface. Even at this time a reaction may set in, but in the large majority of cases death results in from twenty-four to forty-eight hours from exhaustion. The end may be characterized by the symptoms of spurious hydrocephalus—delirium, stupor, convulsions, and coma. As these nervous phenomena are unassociated with any cerebral lesion, they are probably toxemic.

Prognosis.—The prognosis is very bad. The outlook is more favorable when the child has survived the severe symptoms of the first two days. Recovery is always tedious.

Treatment.—The stomach should be washed out with warm water, and the bowel irrigated with cold water.

At first nothing should be given by the mouth except sterilized ice-cold water and iced brandy or champagne. When the stomach is wholly unretentive, stimulants should be given hypodermically. Hot packs (101.4° F.) are very useful in combating collapse. In urgent cases normal salt solution (40 grains to the pint) should be used subcutaneously, from 2 to 3 ounces being injected three or four times daily. If vomiting and purging still continue, small doses of morphin and atropin should be administered hypodermically. Holt gives $\frac{1}{100}$ of a grain of morphin with $\frac{1}{800}$ of a grain of atropin for a child one year old, and repeats the dose, if necessary.

After vomiting has ceased, barley-water, albumin-water, and fresh beef-juice may be given by the mouth. Milk feeding should always be resumed very gradually.

DYSENTERY.

Definition.—An acute or chronic inflammatory disease of the colon, manifested clinically by abdominal pain, tenesmus, and the frequent passage of small stools containing mucus and blood.

Varieties.—(1) Catarrhal; (2) amebic; (3) bacillary.

Etiology.—(1) Warm climates and warm weather; (2) bad hygienic surroundings; (3) ingestion of irritating food; (4) exposure to cold and wet; (5) cachectic states—are the predisposing factors.

The catarrhal form is usually sporadic and appears to have no specific etiology. It is common in temperate zones.

The amebic form is due to the Amœba coli, an organism from three to five times the size of a red blood-cell, consisting of a central mass of granular protoplasm surrounded by a narrow rim of clear protoplasm. It may be endemic or sporadic.

In the bacillary form the pathogenic agent is the *bacillus of Shiga*, a motile, flagellate rod belonging to the colon-typhoid group of bacilli and possessing pronounced agglutinating properties. Bacillary dysentery is commonly epidemic, though it may be sporadic.

While the amebic and bacillary forms are frequently encountered in temperate climates, they are especially prevalent in the tropics.

In the majority of instances dysentery is a water-borne disease.

Pathology.—In the catarrhal form the mucous membrane of the colon is red, swollen, and edematous. Follicular ulceration is sometimes seen.

The amebic form is marked by great swelling and infiltration of the mucosa and serpiginous ulcers with irregular outlines and undermined edges. Abscess of the liver occurs in about 20 per cent. of the cases. In bacillary dysentery the inflammation is often diphtheritic. Ulcers are also found, but, unlike those of the amebic form, they begin in the mucosa and extend regularly into the deeper coats. The bacillus is found with great difficulty in the chronic cases. Abscess of the liver is uncommon.

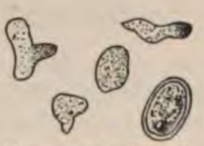

FIG. 2.—Amœba coli.

Symptoms. — *Catarrhal Dysentery.* — Moderate fever (101°–103° F.), abdominal pain, tenderness over the colon, constant desire to defecate, prostration, tenesmus, and the passage of numerous small stools containing mucus and blood. Recovery usually follows in from a week to ten days. The disease occasionally becomes chronic. Complications are rare.

Amebic Dysentery.—In this form the onset is more gradual, fever is not so high, tenesmus is less marked, and the discharges contain living amebæ. In favorable cases the symptoms abate in from six to twelve weeks. In the majority of cases, however, the course of the disease is essentially chronic, and marked by periodic recrudescences.

The chief features of the *chronic form* are dull abdominal pain, tenderness over the colon, frequent stools consisting of scybalous masses covered with tenacious mucus and perhaps with blood and pus. Slight tenesmus, and ultimately extreme anemia and emaciation. Death usually results from complications, the most common of which is abscess of the liver.

Bacillary Dysentery.—The symptoms begin acutely, and are often severe. In addition to mucus and blood, the stools may contain false membrane and gangrenous tissue. Tympanites is common. Typhoid symptoms—delirium, stupor, subsultus tendinum, etc.—are also frequent. Asthenia and emaciation rapidly develop. The blood-serum yields an agglutination reaction with the Shiga bacillus. In some epidemics the mortality has reached 40 per cent. Death usually results from exhaustion or collapse, and very rarely from abscess of the liver or other complications. Occasionally the disease loses its acute character and becomes chronic.

Complications and Sequels.—Hepatic abscess is the most common complication. It occurs chiefly in the amebic form. Peritonitis from perforation or from extension of the inflammation, hemorrhage, multiple neuritis, and intestinal stricture are rare accidents.

Diagnosis.—**Acute Catarrhal Enteritis.**—In this disease tenesmus is absent; the stools are not bloody and mucoid, and are neither so frequent nor so scanty.

Intussusception.—The abrupt onset, persistent vomiting, and presence of a sausage-shaped tumor in the abdomen will generally make the diagnosis clear.

Treatment.—Rest in bed is imperative. In acute cases the diet should be liquid—milk with lime-water, animal broths, and egg-white. In chronic cases soft-boiled eggs, pulled bread, steamed rice, oysters, and tender meats may be allowed.

The stools should be immediately disinfected.

An unirritating purgative (Epsom salts or calomel) is nearly always indicated at the onset. Subsequently, opium should be given to check peristalsis and to relieve tenesmus. It may be given hypodermically in the form of morphin, or by the bowel in form of starch-water (½ fluidounce) and laudanum (10 to 20 drops) injections. Turpentine stupes or sinapisms afford relief. Persistent tenesmus is sometimes controlled by ice suppositories or iodoform suppositories (2 to 5 grains). Internally, bismuth subnitrate (30 to 40 grains), with antiseptics like beta-naphthol-bismuth (10 grains), salol (5 grains), or benzonaphthol (5 to 10 grains), is useful. The following combination is often of value:

℞. Pulveris ipecacuanhæ et opii	ʒss
Bismuthi subnitratis	ʒss
Bismuthi beta-naphthol	ʒj.—M.

Fiant chartulæ No. xij.
SIG.—One powder every two hours.

In many cases the adynamia is so pronounced that stimulants must be used freely.

Two special methods of treatment have been found efficacious—that by ipecac and that by salines:

Ipecac.—On the first day a single large dose of from 30 to 40 grains of powdered ipecac is given stirred up in water. Subsequently from 10 to 20 grains are given daily for three or four days. To prevent emesis, laudanum (15 to 20 drops) is given twenty minutes in advance of the ipecac. A sinapism is applied to the epigastrium, and absolute quiet is enjoined. A successful issue is indicated by the appearance, usually within twenty-four hours, of a copious black stool.

Salines.—A purgative dose of magnesium sulphate is administered at once, and after the bowel has been thoroughly emptied, small doses (1 dram) are given several times a day, preferably in combination with aromatic sulphuric acid, as in the following formula:

℞. Magnesii sulphatis	℥iss
Acidi sulphurici aromatici	f℥ij
Aquæ cinnamomi	q. s. ad f℥vj.—M.

SIG.—A tablespoonful four times a day.

This treatment should be continued for several days after the stools have ceased to be dysenteric.

Chronic Dysentery.—Bismuth subnitrate (½ dram) with intestinal antiseptics is of value. Stengel and others have found sulphur very serviceable in amebic dysentery. Ten grains should be given three or four times a day, combined with a small amount of opium.

Intestinal irrigation with solutions of silver nitrate (10 to 30 grains to 1 pint of cold water) is of the greatest benefit. These injections should be given two or three times a week, the fluid being introduced very gently and slowly by means of a fountain-syringe. When the rectum is very irritable, it is advisable to inject a small quantity of cocain solution (4 per cent.) before introducing the irrigator. In amebic dysentery warm injections of quinin (1 : 5000 to 1 : 2000), creolin (½–1 per cent. mixtures), and benzoyl-acetyl peroxid (1 : 1000) have also been found efficacious.

CHOLERA MORBUS.

(**English Cholera; Cholera Nostras.**)

Definition.—An acute sporadic disease, resembling Asiatic cholera, but not excited by the comma bacillus of Koch.

Etiology.—The summer season predisposes, and irritating food, as unripe fruit, and a sudden change of temperature are the usual exciting causes. An organism resembling the bacillus of true cholera is often present in the discharges.

Symptoms.—There are intense cramps in the stomach, vomiting and purging of bilious material, thirst, moderate fever, and great prostration. In severe cases the discharges become serous, and symptoms of collapse develop.

Diagnosis.—**Asiatic Cholera.**—In this disease there is often no history of dietetic indiscretions; etiologic relation with another case can usually be established; the stools have a characteristic "rice-water" appearance; there are painful cramps in the legs; and the typical cholera bacillus is present.

Mineral Poisoning.—The history, burning pain in the gullet and rectum, erosion of the mouth and throat, and mucous, bloody discharges will usually lead to a correct diagnosis.

Prognosis.—Favorable. Death rarely occurs, except in old or debilitated subjects. The duration is usually from twenty-four to forty-eight hours.

Treatment.—The hypodermic injection of morphin ($\frac{1}{4}$ grain) and atropin ($\frac{1}{100}$ grain) is usually necessary. Hot applications to the abdomen are also useful. Thirst is best relieved by cracked ice. Calomel in fractional doses serves to allay vomiting and to rid the bowel of irritating matter. In many cases an anodyne mixture like the following will act admirably:

℞.	Spiritus camphoræ	f℥ss
	Olei caryophylli	♏xx
	Chloroformi	fʒiij
	Tincturæ opii deodorati	
	Tincturæ capsici	aa fʒij.—M.

Sɪɢ.—Shake well. Thirty to forty drops in water every half hour to two hours, as required. (H. C. Wood.)

Collapse will require hot baths, diffusible stimulants (ammonia, ether, brandy), and subcutaneous injections of salt solution.

APPENDICITIS.

(**Typhlitis; Perityphlitis.**)

Definition.—An inflammation of the appendix vermiformis.

Pathology.—There are three varieties: Catarrhal, ulcerative, and interstitial.

Catarrhal Appendicitis.—In mild cases the appearances are, no doubt, similar to those observed in catarrh elsewhere, but in severe cases the wall of the appendix is infiltrated with round-cells, and the mucous membrane is denuded of epithelium and presents a granular surface. This latter condition may eventuate in septic peritonitis, chronic appendicitis with relapses (*recurrent appendicitis*), or union of the granulating surfaces with complete obliteration (*appendicitis obliterans*).

Ulcerative Appendicitis.—In this type the wall of the appendix is the seat of a more or less localized ulcer. It may be associated with the presence of fecal concretion or a foreign body, or it may be the result of typhoid or tubercular infection.

Interstitial Appendicitis.—In this form the wall of the appendix is the seat of a necrosis, which is not infrequently gangrenous. It may be primary, infection taking place through the lymphatics, or secondary to the catarrhal or ulcerative form. It terminates in perforation, thereby exciting a most virulent type of peritonitis.

Appendicitis is always due to the action of pathogenic bacteria, the chief offenders being the Bacillus coli communis, Streptococcus pyogenes, Staphylococcus pyogenes aureus, typhoid bacillus, and tubercle bacillus. Of these, the Bacillus coli communis, a natural habitant of the bowel, is most commonly present. Under ordinary conditions it is harmless, but when the circulation of the appendix is interfered with from any cause or the coats of the tube are abraded, infection is liable to arise.

Etiology.—It is more common in males than in females. It is most frequent between the fifteenth and thirtieth years. Exposure, errors in diet, intestinal catarrh, traumatism, and the lodgement in the appendix of fecal concretions or foreign bodies predispose to the disease. It may follow some infection like typhoid fever, influenza, or tuberculosis. It may be induced by twisting of the appendix.

Symptoms.—(1) Sudden pain, often general at first, but later most marked in the right iliac region. (2) Circumscribed tenderness, most frequently detected over McBurney's point—a point midway on a line between the umbilicus and the anterior superior iliac spine. (3) Fever, ranging between 100° and 103° F. (4) Localized rigidity in the right iliac fossa, or the presence of a definite tumor. (5) Dorsal decubitus with the right thigh flexed. (6) Gastro-intestinal disturbances—anorexia, nausea, vomiting, constipation, or rarely diarrhea.

Terminations.—Resolution, general peritonitis, and localized abscess. The location of the abscess depends on the position of the appendix. It may be found in either of the lower quadrants or beneath the diaphragm (subphr abscess). The pus may be discharged through dominal walls, the bowel, bladder, or vagina, or it may escape into the tissues of the lumbar region or thigh. Appendicitis occasionally excites hepatic abscess, the infection being carried through the portal vein.

Diagnosis.—**Typhoid Fever.**—The gradual onset, characteristic temperature-curve, epistaxis, mental hebetude, diarrhea, splenic enlargement, and, later, the rash and Widal reaction will indicate typhoid fever.

Renal Colic.—This may be recognized by the absence of fever and of local rigidity, and the presence of hematuria.

Acute Inflammation of the Gall-bladder.—Pain and tenderness in the right hypochondrium, a smooth, mobile tumor, and a history of biliary colic would suggest this condition.

Tubal Disease.—The history and results of pelvic examination will usually prevent an error in diagnosis.

Prognosis.—The prognosis depends on the type. The average mortality is about 14 per cent.

Treatment.—The patient should be kept in bed at absolute rest. The diet should be restricted to small quantities of bland liquids—milk, albumin-water, and broths. Constipation is best relieved by enemas of warm water. Locally, cold or heat may be applied, according to the sensations of the patient. If the pain is very severe, morphin may be administered hypodermically; only the minimum amount necessary to afford a measure of relief is to be used, however, as by obscuring the symptoms, the drug prevents an accurate study of the progress of the case.

An operation should be urged—(1) At once in all cases in which the onset is very severe, the symptoms indicating special severity being marked right-sided tenderness and rigidity, distention, and vomiting, with or without fever; (2) in cases of moderate severity which manifest no improvement after the lapse of forty-eight hours; and (3) in cases in which the symptoms, after decided improvement, return. On the contrary, operation is rarely required, at least during the attack—(1) In cases of a mild type, in which the pain is unaccompanied by rigidity, distention, nausea, or vomiting; and (2) in cases of moderate severity in which improvement is noticeable within forty-eight hours. Operation during the quiescent stage, when the element of danger is almost entirely removed, is to be recommended—(1) When an acute attack has been followed by persistent tumefaction and tenderness, intestinal disturbances, or impairment of the general health; (2) when there have already been two attacks, even of moderate severity; and (3) when mild attacks occur with such frequency as to induce disability.

INTESTINAL OBSTRUCTION.

(Ileus.)

Intestinal obstruction may be either acute or chronic. The chief causes of the *acute form* are: (1) Strangulation; (2) intussusception; (3) volvulus; (4) impaction of foreign bodies or gall-stones; (5) paresis of the intestine; (6) congenital malformation or stricture.

Chronic obstruction may be due to—(1) Impaction of feces; (2) stricture; (3) tumors of the bowel or of neighboring organs.

Symptoms of Acute Obstruction.—(1) Sudden abdominal pain—at first paroxysmal, but later continuous; (2) constipation, soon becoming absolute; (3) vomiting, persistent and ultimately of a stercoraceous character; (4) abdominal distention; (5) visible peristaltic waves; (6) collapse, indicated by pinched features, sunken eyes, a cold, clammy surface, and a frequent feeble pulse.

Symptoms of Chronic Obstruction.—The symptoms usually develop gradually. Acute symptoms may appear, however, when the occlusion becomes complete. The chief features are intractable constipation, colicky pains, distention of the abdomen, and gradual failure of health. The stools may be ribbon-shaped or in the form of scybalous masses, and are sometimes coated with mucus and blood. Vomiting is not common.

Diagnosis.—Early vomiting, slight distention, suppression of urine, and rapid collapse point to an obstruction *high in the small intestine.*

Acute Generalized Peritonitis.—The history, early appearance of fever and of diffuse tenderness, signs of effusion, and absence of stercoraceous vomiting will indicate peritonitis.

Strangulation.—This often occurs in external hernia, when it can be recognized by an examination of the inguinal, femoral, and umbilical rings.

Internal strangulation is very common. It may be due to the slipping of a coil of intestine under bands of adhesions, the result of a former peritonitis, or under Meckel's diverticulum that is abnormally attached to the abdominal wall, or through a slit in the omentum or mesentery, the foramen of Winslow, or the diaphragm. It usually occurs in young adults; there is often a history of injury or of peritonitis, and the symptoms are very acute.

Intussusception or Invagination.—This is the slipping of a portion of the intestine into the part immediately below it. It occurs especially in children. Its exciting cause is probably irregular peristalsis, whereby one part of the bowel is constricted while the adjoining part is dilated. The usual seat is the ileocecal region.

Multiple invaginations are frequently found postmortem, which have resulted from the irregular peristalsis occurring just before death; they possess no inflammatory characteristics. In invaginations not cadaveric the parts are injected, swollen, and covered with lymph.

The age of the patient, the sudden abdominal pain, the vomiting, the passage with tenesmus of mucus and bloody feces, and the presence of a sausage-shaped tumor in the region of the ascending colon are the diagnostic features. Occasionally the invaginated portion can be felt in the rectum.

Death usually results from gangrene, peritonitis, or collapse. A favorable termination sometimes results from the escape of the incarcerated part, or by a sloughing off of the strangulated portion and adhesion of the serous surfaces.

Volvulus or Twist or Knot of the Bowel.—Volvulus occurs most commonly in middle-aged men. The usual seat is the sigmoid flexure. A relaxed and lengthened mesentery is a predisposing factor. It cannot be recognized with certainty without abdominal section.

Impaction of Foreign Bodies.—Foreign bodies swallowed by accident or design, gall-stones, or enteroliths may cause acute intestinal obstruction. The history may aid in the diagnosis.

Gall-stone ileus is most frequently met with in women after the fiftieth year. The ileocecal region is the usual seat of the obstruction.

Paresis of the Bowel.—This occasionally develops idiopathically in nervous women. It may also result from peritonitis, an abdominal operation, the reduction of a hernia, or traumatism.

Congenital Malformation.—This rare form of obstruction usually consists in an imperforate condition of the anus or rectum. It may be recognized by digital examination.

Impaction of Feces.—This may occur at any age, but it is most often seen in persons past middle life. The usual seat of the impaction is the rectum or colon. The condition may be recognized by the gradual onset of the symptoms, the history of habitual constipation, and by the presence of a fecal mass in the rectum or of an irregular, painless, doughy tumor in the region of the colon.

Stricture and Tumors.—Cicatricial contraction may result from syphilitic, tuberculous, or dysenteric ulceration. The rectum is the part most frequently involved. The most common tumor of the bowel is cancer. It is usually seated in the rectum. The diagnosis may be established by the history of the case, the gradual onset of obstructive symptoms, impairment of health, painful defecation, the size and form of the stools, the presence of blood and pus in the stools, and the results of a physical examination.

Treatment.—*Acute Obstruction.*—Food by the mouth should be withheld. Ice may be given to quench thirst. Nutritive enemata should be employed in the weak. *Cathartics are contraindicated.* Pain is best relieved by warm applications and the administration of morphin hypodermically. Washing out the stomach three or four times daily is recommended for the persistent vomiting. Distention of the large bowel with warm water or gas should be practised in doubtful cases and intussusception. It is best done under anesthesia with the patient in knee-elbow position. After failure with these methods operation should not be delayed; the earlier its performance, the greater the chance of success.

Chronic Obstruction.—The treatment will vary with the cause. Surgical interference is frequently required.

In fecal impaction injections of warm water, of oil (4 to 6 fluidounces), or of aqueous solutions of ox-gall (2 drams to 1 pint) are efficient. Salines may be administered by the mouth. Massage is sometimes useful. Hard rectal accumulations may have to be removed by the fingers or a suitable scoop.

ANIMAL PARASITES.

CESTODES OR TAPE-WORMS.

Varieties.—Tænia solium; Tænia saginata; Bothriocephalus latus; Tænia echinococcus.

Life History.—The eggs of the tape-worm are ingested by animals (the intermediary host), and embryos or proscolices are liberated in the stomach; these migrate to the muscles or organs, where they become transformed into encysted larvæ or scolices. The encysted larvæ are known as cysticerci or "measles." When flesh infested with cysticerci is eaten by man (the host), the scolex is liberated, fastens itself to the mucous membrane of the bowel, and rapidly develops into a mature worm.

Tænia Solium.—This worm exists in the larval state in the hog. The mature worm is two or three yards in length. Its head, which is the size of a pin-head, is provided with four pigmented, cup-like suckers, surrounded by a double row of hooklets, and is attached to the body by a thread-like neck. The sexual orifice is in the center of the broad surface of the segment. This parasite is rare in America.

Tænia Saginata or Mediocanellata.—The larval form occurs in the ox. The mature worm is five or six yards in length. The head is larger than that of the Tænia solium, and has four large suckers, but no hooklets. The segments are fatter, and the uterine branches are finer and more numerous than in the Tænia solium. It is the common tapeworm of this country.

Bothriocephalus Latus.—The intermediate host is some form of fish (pike). The adult worm is from five to ten yards in length. The head is flattened and club-shaped, presents two groove-like suckers, but is without hooklets. This worm is frequent in certain parts of Europe, but it is rare in America.

Tænia Echinococcus.—This worm in its adult form occurs in the intestine of the dog; in man it appears only in the larval condition (see Hydatid Cysts of the Liver).

Symptoms.—In many cases there are no subjective symptoms. Some patients, however, present the symptoms of dyspepsia, bulimia, colicky pains, progressive emaciation, anemia, and certain reflex manifestations—vertigo, palpitation, itching of the nose, spasms, and choreic movements. The diagnosis rests on the discovery of the tenia segments or eggs in the stools.

The bothriocephalus may produce a very severe anemia, which has been ascribed to the secretion of substances having a destructive action on the red blood-cells.

Treatment.—By way of preparatory treatment it is advisable to restrict the diet for a day or two to liquids and to empty the bowel as completely as possible by saline purges. The best anthelmintics are oleoresin of aspidium (½ to 1 dram), pumpkin seed (2 to 3 ounces), and pelletierin (5 to 8 grains).

℞. Oleoresinæ aspidii . . . . f℥ss
Pulveris acaciæ et sacchari . . āā q. s.
Aquæ cinnamomi . . . . . . q. s. ad f℥ij.—M.
SIG.—One tablespoonful, to be repeated if necessary.

A purge should be given a few hours after the anthelmintic. The treatment is successful only when the head of the worm is passed.

NEMATODES OR ROUND-WORMS.

Ascaris Lumbricoides (*Common Round-worm*).—Round-worms develop from eggs which have entered the body through water or food. They are of a brownish or pinkish color, and in form resemble earth-worms. They occupy the small intestines, but occasionally migrate, entering the stomach, bile-ducts, and even the larynx. They are most commonly found in children.

Symptoms.—Often absent. Sometimes there are dyspepsia, mucous stools, colicky pains, voracious appetite, anemia, and reflex nervous phenomena—night-terrors, grinding of the teeth, pruritus of nose and anus, choreic movements, and convulsions.

Treatment.—Santonin ($\frac{1}{4}$–$\frac{1}{2}$ grain); wormseed oil (10 drops in capsule or on sugar); fluid extract of spigelia (1 to 3 fluidrams) are efficient remedies. The anthelmintic should be followed by a purge.

℞. Santonini	gr. vj
Hydrargyri chloridi mitis	gr. vj
Sacchari	gr. xxiv.—M.

Fiant in chartulæ No. xij.
SIG.—One powder morning and evening. (STARR.)

Oxyuris Vermicularis (*Seat-worm; Pin-worm*).—Pin-worms are from one-eighth to one-half inch in length. They are most commonly seen in children, infection probably taking place through water or green vegetables. They occupy the rectum and colon, and are often present in great numbers. They produce intense itching, particularly at night.

Treatment.—Copious injections of a cold infusion of quassia (1 ounce to 1 pint) or of a solution of sodium chlorid (1 dram to 1 pint) usually prove successful. In obstinate cases anthelmintics (santonin or chenopodium) should be given by the mouth. Care should be taken to prevent reinfection with the eggs, which are produced in large numbers.

Uncinaria Duodenalis (Ankylostoma Duodenale) and Uncinaria Americana.—These are hook-worms of the genus *Uncinaria*, measuring from 8 to 16 mm. in length. The former prevails in the old world and the latter in America. Their habitat is the small intestine.

Uncinariasis or ankylostomiasis is a common disease in tropical and subtropical countries. In temperate regions it prevails chiefly among miners. It is believed that the parasites may enter the body either through the medium of drinking water or directly through the skin. Anemia, more or less severe, is the chief symptom. There is nearly always a well-marked eosinophilia. Digestive disturbances may also be present. The diagnosis rests upon the discovery of the ova in the stools.

Treatment.—Thymol, in doses of 30 grains, repeated in two hours, and followed by a purgative, is almost a specific.

Filaria Sanguinis Hominis.—A small thread-like worm, most commonly seen in warm climates. The adult occupies the lymphatics, and the female brings forth a great number of actively motile embryos, which soon find their way into the blood-current. It is a curious fact that the embryos are found in the blood only at night. The medium of infection is the mosquito, which probably carries the embryo from the blood to the drinking-water.

Symptoms.—Chyluria, hematuria, elephantiasis, and lymph-scrotum may result from occlusion of the lymphatic vessels.

Treatment.—Thymol (3 to 5 grains) and methylene-blue (1 to 3 grains) have been used, but they are rarely efficacious.

Trichina Spiralis.—This is a small worm derived from the hog. Man is infected by eating insufficiently cooked pork containing the encapsulated larvæ. The worm is set free in the stomach, where it develops and brings forth living embryos in immense numbers. These, entering the bowel, soon migrate into the muscles, where they develop, coil themselves up, and become encapsulated. Trichinous capsules, impregnated with lime-salts, are visible to the naked eye, and are sometimes detected accidentally at autopsies.

Symptoms of Trichiniasis (Trichinosis).—No decided symptoms develop unless the parasites have been ingested in large numbers. In well-marked cases gastro-intestinal disturbances appear on the second or third day. These disturbances consist in colicky pains, nausea, vomiting, and serous diarrhea.

In from one to two weeks symptoms of an acute myositis develop, characterized by severe muscular pains and soreness, edema, beginning in the face, sweating, and fever. Hoarseness and dyspnea may occur from involvement of the larynx and diaphragm.

Leukocytosis is marked, the eosinophilic cells being especially increased. In certain cases the symptoms closely resemble those of typhoid fever. In favorable cases recovery is effected in from two to eight weeks.

Diagnosis.—**Typhoid Fever.**—The history, the presence of eosinophilia, of intense muscular soreness, of edema, of parasites in the stools or in a fragment of muscle removed from the arm, and the absence of a typical rash and of the Widal reaction will lead to a correct diagnosis.

Muscular Rheumatism.—The history, the presence of gastro-intestinal symptoms, of edema, and of eosinophilia will suggest trichiniasis.

Prognosis.—This depends upon the number of worms ingested. Early diarrhea is favorable. The mortality ranges from 5 to 30 per cent.

Treatment.—The most efficient prophylactic measure is the thorough cooking of all pork products. In the first stage cathartics are indicated. Anthelmintics—santonin, aspidium, and thymol—have been recommended. After migration, the indications are to relieve pain by means of opiates, hot baths, and warm embrocations, and to support the strength by concentrated liquid diet and stimulants.

DISEASES OF THE PANCREAS.

HEMORRHAGE INTO THE PANCREAS.

Etiology.—Hemorrhage into the pancreas may result from traumatism. It may be due to passive congestion, to hemorrhagic diseases (scurvy, purpura, etc.), or to acute infections. It is very commonly associated with organic disease of the pancreas—acute pancreatitis, arteriosclerosis, cysts, and cancer.

Symptoms.—Sudden severe pain in the epigastrium, vomiting, tympanites, dyspnea, and collapse are the chief symptoms. The diagnosis can rarely be made with certainty.

Prognosis.—Most cases prove fatal within twenty-four or thirty-six hours, death being due to an arrest of the heart from injury to the celiac plexus (Zenker) or semilunar ganglion (Friedreich). Pancreatitis, cyst of the pancreas, and peritonitis are possible terminations. Complete recovery is rare.

Treatment.—Morphin is required for the pain, and stimulants for the collapse. If the patient survives the initial collapse and symptoms of suppuration develop, operation is indicated.

ACUTE PANCREATITIS.

Varieties.—Hemorrhagic, suppurative, and gangrenous.

Etiology.—(1) This may result from gall-stone impaction, bile being retrojected into the pancreatic duct; (2) from inflammatory affections in neighboring parts—gastroduodenal catarrh, gastric ulcer, or cancer; (3) from general infections—specific fevers and pyemia; (4) from traumatism. The immediate cause is bacterial infection.

Pathology.—In the hemorrhagic form the organ is irregularly enlarged and the seat of hemorrhagic extravasation. Opaque, white spots of a tallowy consistence are frequently found in the interlobular tissue, omentum, and surrounding parts, and represent areas of *fat necrosis.*

In suppurative pancreatitis there may be multiple abscesses or one large collection of pus. More or less extensive areas of necrosis are found. Thrombosis of the portal and splenic veins is frequently encountered. Pancreatic abscesses may become encapsúlated or they may rupture into the peritoneum, stomach, or duodenum.

Gangrenous pancreatitis is usually secondary to one of the other varieties.

Symptoms.—The chief symptoms are sudden intense pain in the epigastrium, distention of the epigastrium, vomiting and collapse, followed in suppurative cases by irregular fever, constipation, slight jaundice, delirium, and rapid loss of weight.

Diagnosis.—**Intestinal Obstruction.**—In this condition the onset is usually less severe, fecal vomiting is common, pain and distention are less frequently limited to the epigastrium, and constipation is absolute, not even flatus beingpassed.

The history will sometimes serve to differentiate the condition from *biliary colic*, *perforating gastric ulcer*, and the effects of an *irritant poison*.

Prognosis.—Very unfavorable. The duration varies from a day or two in the hemorrhagic form, to several weeks in the chronic suppurative variety. Recovery may follow operation or rupture of the abscess into the bowel. It may rarely end in chronic pancreatitis.

Treatment.—Operation after the initial collapse offers some hope of cure.

CHRONIC PANCREATITIS.

(Cirrhosis of the Pancreas.)

Etiology.—It may result—(1) From closure of the pancreatic duct by gall-stones impacted in the common bile-duct; (2) from extension of inflammation in gastroduodenal catarrh or pyloric ulcer; (3) from syphilis or alcoholism; (4) from sclerosis of the pancreatic arteries, and, possibly, (5) from acute pancreatitis.

Pathology.—The chief lesions are an overgrowth of the fibrous tissue and more or less degeneration or atrophy of the cellular elements.

Symptoms.—The symptoms are obscure. Flatulent dyspepsia, paroxysmal epigastric pain, a tendency to diarrhea, and slight jaundice are the usual features. Albuminuria and glycosuria may occur. Fatty stools have been noted in a few instances. When the islands of Langerhans are involved in the degenerative process, the symptoms of *diabetes mellitus* develop.

Prognosis.—The disease runs a slow course. If glycosuria develops, the outlook is more grave.

Treatment.—The use of fats and starches should be restricted. Carbonated waters are said to increase pancreatic secretion. Pancreatin is recommended. Surgical treatment offers a good chance of recovery in gall-stone cases.

CANCER OF THE PANCREAS.

Etiology.—The disease most frequently occurs in males past forty years of age.

Pathology.—Pancreatic cancer is usually primary; it generally involves the head of the gland, and is commonly of the scirrhous variety.

Symptoms.—These include disturbances of digestion, rapid loss of flesh and strength, anemia, intense deep-seated epigastric pain, and the presence of a tumor. The latter is usually found a little above the navel; it is but slightly movable, deep seated, and often pulsatile from its relation to the aorta. The pain often occurs in paroxysms, especially at night, and may be associated with the symptoms of collapse. Progressively increasing jaundice, with enlargement of the gall-bladder, is a frequent symptom, and results from the pressure of the tumor upon the common bile-duct. Pressure on the portal vein may cause ascites. Glycosuria is an occasional symptom. The stools rarely contain free fat, but the presence of abundant undigested muscular fibers in the dejections in the absence of diarrhea is, according to Fitz, highly suggestive.

Diagnosis.—Gastric cancer. In this condition the tumor is more freely movable, is usually associated with dilatation of the stomach and with marked gastric symptoms. Pain is not usually so severe. Jaundice is rare.

CYSTS OF THE PANCREAS.

Varieties.—(1) Retention cysts from impaction of a calculus, stricture, or tumor; (2) apoplectic cysts from hemorrhagic extravasation; (3) hydatid cysts; (4) congenital cysts; (5) proliferation cysts (carcinomatous or adenomatous).

Pathology.—Pancreatic cysts may be single or multiple. They lie behind the stomach, and may contain from a few ounces to several gallons of a grayish or brownish, viscid fluid, of an alkaline reaction, of a specific gravity between 1010 and 1024, and presenting the characteristics of pancreatic secretion.

Symptoms.—These are very variable, the most common being epigastric pain, vomiting, constipation, or diarrhea, disturbances of digestion, loss of flesh, and occasionally intestinal hemorrhage. Free fat and much undigested muscular fiber may be found in the stools and sugar in the urine. Physical examination often reveals in the upper part of the abdomen a smooth, elastic, fluctuating tumor which on aspiration yields a fluid capable of emulsifying fats, of converting starch into sugar, and of digesting albumin.

Prognosis and Treatment.—The prognosis is guardedly favorable under operative treatment.

PANCREATIC CALCULI.

Pancreatic calculi are probably due to altered glandular secretion or infection. Their passage through the duct excites *pancreatic colic*, the symptoms of which resemble biliary colic, but the pain is more apt to radiate to the left and is unattended with jaundice. The coexistence of glycosuria with fatty stools, and the discovery in the stools of concretions containing chiefly carbonate or phosphate of lime, would confirm the diagnosis.

DISEASES OF THE LIVER.

The *liver* is situated in the right hypochondrium, with a small part projecting through the epigastrium to the left hypochondrium.

Area of Liver Dulness.—The absolute dulness (part uncovered by lung) extends in the mammary line from the upper border of the sixth rib to the costal margin; in the axillary line, from the seventh rib to the eleventh rib; in the scapular line, from the ninth rib to the eleventh rib; in the median line, the upper border is lost in the cardiac dulness, while the lower border lies midway between the ensiform cartilage and the umbilicus. Slight dulness in the mammary line begins at the fifth rib.

Palpation.—*Palpation* of the liver is practised to determine position, size, form, and consistence and to detect any tenderness or pulsation.

Conditions in which the liver is palpable:

1. In thin subjects the edge is sometimes palpable under normal conditions.

2. In very young children in whom the liver is always proportionately large.
3. In depression of the liver, as by a pleural effusion or by a consolidated lung.
4. When the suspensory ligaments are relaxed and the liver "wanders."
5. In enlargement of the organ from any cause.
6. In certain abnormalities of form, as in the "corset liver."

Superficial Irregularities.—Small irregularities may be noted in cancer of the liver, syphilis of the liver, and very rarely in atrophic cirrhosis.

Large prominences are sometimes noted in tumors, abscesses, and hydatid cysts.

Consistence.—The liver is firm to the touch in hypertrophic cirrhosis, cancer, congestion, leukemic infiltration, and amyloid disease. In abscess and hydatid disease the resistance is less marked and sometimes fluctuation can be noted.

Tenderness.—The liver is tender in congestion, abscess, cancer, hypertrophic cirrhosis, and in affections complicated with perihepatitis.

Pulsation may be detected in the venous congestion resulting from tricuspid regurgitation, in abdominal aneurysm, and in tumors of the left lobe resting on the aorta.

Percussion.—Percussion determines size and resistance.

The liver is uniformly enlarged in: (1) Congestion, active and passive. (2) Fatty infiltration. (3) Amyloid infiltration. (4) Hypertrophic cirrhosis. (5) Leukemic infiltration. (6) Infiltrating carcinoma.

Irregular enlargements of the liver are noted in: (1) Cancer. (2) Abscess. (3) Hydatid disease. (4) Syphilis.

The liver is diminished in size in: (1) Atrophic cirrhosis, late stage. (2) Fatty degeneration. (3) Acute yellow atrophy. (4) Senile atrophy. The area of hepatic dulness may be diminished from certain extrinsic causes, namely, pulmonary emphysema, excessive tympanites, and perforation of the stomach or bowel.

JAUNDICE OR ICTERUS.

Definition.—Pigmentation of the tissues and secretions with bile-pigments.

Varieties.—(1) Obstructive jaundice. (2) Toxemic jaundice.

Etiology of Obstructive Jaundice.—Obstruction to the outflow of bile leads to its accumulation and reabsorption.

Obstruction may be due to the following causes:

1. Stricture of the bile-duct, congenital or acquired.
2. Catarrh of the bile-ducts or of the duodenal mucous membrane around the orifice of the ductus choledochus.
3. Foreign bodies in the ducts, as gall-stones or parasites.
4. Tumors of the liver or of adjacent viscera compressing the ducts. Fecal accumulations, a pregnant uterus, and displaced organs may similarly compress the ducts.
5. Spasm of the bile-ducts. This has been advanced as the cause of the jaundice that occasionally follows emotional excitement.

Symptoms.—The skin, mucous membranes, and secretions are stained yellow. The discoloration is usually first noticed in the conjunctivæ. The stools are light, the urine is dark, and in bad cases resembles porter. The pulse is usually slow, and the temperature slightly subnormal. There is often more or less mental depression, and in chronic cases delirium, convulsions, and coma occasionally develop. Itching of the skin is often noted, and urticaria is a common complication. In grave cases subcutaneous ecchymoses may appear.

Diagnosis.—Other discolorations, like the bronze hue of Addison's disease and the green tint of chlorosis may resemble jaundice, but in these cases the conjunctiva remains white and the urine lacks bile.

Etiology of Toxemic Jaundice.—This form of jaundice may result from—(1) Certain poisons, as toluylendiamin, phosphorus, anilin, chloroform, and snake venom. (2) Certain general diseases of infective or toxic origin, as septicemia, acute yellow atrophy, malaria, relapsing fever, and pernicious anemia.

In toxemic jaundice there is also obstruction, but it is situated in the minute bile-ducts instead of in the large ones. The chief cause of the obstruction is probably catarrhal inflammation (cholangitis), excited by the poisons circulating in the blood, although extensive destruction of the red blood-cells, which leads to the secretion of a thick, viscid bile, rich in pigments (polychromia), may be a contributing factor in some cases.

Symptoms.—These are much the same as in obstructive jaundice, but the staining of the skin is not so intense, the stools still contain bile, and the constitutional symptoms are apt to be more severe.

ICTERUS NEONATORUM.

Physiologic icterus in the new-born is slight; according to Quincke, it is probably due to the passage of blood rich in bile-pigments absorbed from the bowel directly into the vena cava by way of the ductus arantii, which remains patent for several days after birth.

Pathologic icterus in the new-born is marked, and commonly proves fatal. It results from congenital stricture of the common bile-duct, syphilis of the liver, or septic infection through the umbilical vein.

CHOLEMIA.

(Cholesteremia; Acholia; Hepatargy.)

The term cholemia has been applied to a group of symptoms which sometimes arises in chronic jaundice and in diseases of the liver characterized by extensive destruction of the liver-cells, such as acute yellow atrophy, cirrhosis, and cancer.

The **symptoms** of cholemia include delirium, convulsions, stupor, and coma. Subcutaneous ecchymoses and hemorrhages from mucous membranes are also frequently observed.

The cause of this condition is supposed to be the retention in the blood of toxic matters which the liver normally converts or eliminates.

CATARRHAL JAUNDICE.

(Catarrhal Hepatitis; Catarrhal Angiocholitis; Catarrh of the Bile-ducts.)

Ætiology.—(1) The most common cause is the extension of a gastroduodenal catarrh into the ducts. (2) Primary inflammation of the ducts may result from exposure to cold and wet. (3) It may be induced by irritation from gall-stones. (4) It may be infectious, complicating malaria, pneumonia, relapsing fever, and similar diseases.

Pathology.—The large ducts are particularly affected; the mucous membrane is swollen and covered with tenacious mucus. When the gall-bladder is compressed, bile is ejected through the duodenal orifice with less ease than is natural.

Symptoms.—(1) Symptoms of gastroduodenal catarrh usually precede. These are: Coated tongue, anorexia, fetid breath, epigastric distress, vomiting, and perhaps diarrhea. (2) Obstructive jaundice, indicated by yellow skin and conjunctivæ, light stools, and dark urine, is a constant symptom. (3) In acute cases there is slight fever with swelling and tenderness of the liver.

Diagnosis.—This is based upon the acute course, the mild character of the symptoms, the history of preceding gastric catarrh, and the youth of the patient.

Prognosis.—Favorable. It rarely becomes chronic. The average duration of the disease is from two to six weeks.

Treatment.—The diet should be simple and digestible. Fatty and saccharine food should be avoided. Milk, broths, eggs, lean meats, oysters, and well-cooked cereals are admissible. Sodium phosphate (1 dram three times a day), silver nitrate (¼ grain three times a day), and ammonium chlorid (5 to 10 grains three times a day) are of value in relieving the primary gastroduodenal catarrh. In obstinate cases nitrohydrochloric acid may prove beneficial. Daily irrigation of the colon with from 1 to 2 quarts of cold water is sometimes of service. Free water-drinking between meals is to be recommended. Alkaline mineral waters (Vichy, Vals, Hathorn) often act well.

Chronic catarrhal jaundice may follow repeated acute attacks; in the large majority of cases, however, it is a sequel of stenosis of the common bile-duct from gall-stones, stricture, or pressure from without. A constant symptom is chronic jaundice. In some cases there are recurrent attacks of intermittent fever with chills and sweating (Charcot's intermittent hepatic fever).

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ACUTE CHOLECYSTITIS. g.

Definition.—Acute inflammation of the gall-bladder.

Etiology.—The disease is always infectious, the organisms most commonly present being the colon bacillus, typhoid bacillus, pneumococcus, staphylococcus, and streptococcus. Injury to the mucosa by gall-stones is an important predisposing factor. It is not an uncommon sequel of typhoid fever and pneumonia.

Pathology.—The inflammation may be catarrhal or suppurative. Suppurative cholecystitis (*empyema of the gall-bladder*) is usually associated with purulent inflammation of the bile-ducts, and, unless promptly relieved by operation, proceeds to ulceration or gangrene and general peritonitis.

Symptoms.—In catarrhal cases the symptoms are slight fever, pain in the hepatic region, tenderness and enlargement of the gall-bladder, and, occasionally, jaundice. In the suppurative form there are severe paroxysmal pain, a septic type of fever, leukocytosis, enlargement and tenderness of the gall-bladder, and, in some cases, jaundice.

Diagnosis.—It must be distinguished from *appendicitis*, *subphrenic abscess*, and *acute pancreatitis*. The discriminating features are the history of previous cholelithiasis, typhoid fever, or pneumonia, and the locality of the pain, tenderness, and swelling.

Prognosis.—Very grave in suppurative cases. Early operation, however, offers considerable hope of success.

8

CHOLELITHIASIS.

(Gall-stones; Biliary Calculi.)

Etiology.—Gall-stones are three or four times more common in women than in men. They occur most frequently after middle life, and are rarely seen before twenty-five. Sedentary habits, high living, tight lacing, obstruction of the ducts, and other factors that favor stagnation and inspissation of the bile predispose to their formation. Their occurrence after typhoid fever and other infections is not uncommon. The direct cause appears to be a microbic infection of the gall-bladder, in consequence of which excessive quantities of cholesterin and lime are excreted by the irritated mucous membrane and deposited upon desquamated epithelium or clumps of bacteria.

Pathology.—Gall-stones may be found in the ducts, but in the large majority of cases they originate in the gall-bladder. There may be one or several hundred. When multiple, they are found with facets, from attrition. The size varies from that of a grain of sand to that of a large walnut. The color varies from light yellow to dark green. 107, —The chief constituent is cholesterin, but bile-pigments and lime-salts also enter in their composition. On section, they usually present a concentric arrangement.

Events.—(1) Gall-stones often remain quiescent in the gall-bladder. (2) In consequence of violent expulsive efforts, excited by irritation of the gall-bladder, they may be extruded into the bowel, intense pain (*biliary colic*) marking their passage through the ducts. (3) Instead of making a complete exit, they may slip back into the gall-bladder or they may become impacted in the cystic duct, or, more often, in the lower part of the common duct. (4) They may perforate into the duodenum, peritoneum, lung, stomach, or kidney, or externally. Perforation may be followed by stricture of the ducts or by fistulous communications between the ducts and the gastro-intestinal canal. Perforation into the duodenum is not a rare cause of intestinal obstruction. (6) Invasion of the gall-bladder with pathogenic microbes in cases of cholelithiasis is not infrequently followed by suppurative cholecystitis, suppurative angiocholitis, and abscess of the liver. (7) The prolonged irritation by calculi may ultimately give rise to carcinoma of the biliary passages.

Symptoms of Biliary Colic.—(1) The attacks begin abruptly with intense pain radiating from the hypochondriac region to the right shoulder. There are usually tenderness and rigidity over the gall-bladder. Chill and fever (102°–103° F.) often mark the onset. The symptoms of intense pain are obvious—anxious face, cold sweat, feeble pulse, and vomiting. Jaundice may follow from obstruction, but it is often absent. If the stone escapes, it may subsequently be found in the stools. The attack may last from a few hours to several days.

Diagnosis.—**Renal Colic.**—In this affection the pain radiates from the lumbar region along the ureter into the bladder and genitals. Frequent micturition is a common symptom. There is no jaundice. Blood or the stone may be found in the urine.

Intestinal colic produces pain that radiates around the umbilicus. There are flatulence and borborygmi. Jaundice is absent.

Gastralgia.—Pain is over the whole stomach, does not radiate to the shoulder, and is relieved by pressure. There is no jaundice.

Gastric Ulcer.—Pain is closely related to eating. There are localized tenderness in the epigastrium, hyperacidity, and frequently hematemesis.

Symptoms of Obstruction of the Cystic Duct.—Obstruction of the cystic duct may be followed by cholecystitis, by atrophy of the gall-bladder, or by dropsy of the gall-bladder (*hydrops vesicæ felleæ*). In the last condition the gall-bladder can often be felt as a pear-shaped, elastic, movable tumor, projecting from the lower margin of the liver. Jaundice is not present, and subjective symptoms are slight.

Symptoms of Obstruction of the Common Duct.—In typical cases the symptoms are—(1) Chronic jaundice showing marked variations in intensity; (2) pain, also subject to distinct exacerbations; (3) recurrent attacks of intermittent fever, with chills and sweats (Charcot's hepatic fever). The liver is not enlarged; the gall-bladder is not distended, but often atrophied from antecedent attacks of cholecystitis. This condition may last for months or years. It not infrequently leads to suppurative angiocholitis, to biliary cirrhosis, or to acute or chronic pancreatitis.

Diagnosis.—**Obstruction of the Common Duct from Without (Cancer).**—The jaundice increases steadily and is without remission, the gall-bladder is enlarged, and characteristic colic and hepatic fever are wanting.

Prognosis.—In the absence of complications the prognosis of cholelithiasis is good. It must be borne in mind, however, that grave complications (suppurative cholecystitis or angiocholitis, perforation, hemorrhagic pancreatitis) may arise most unexpectedly.

Treatment.—Efforts must be directed to keeping the stones quiescent by preventing irritation or catarrh of the gall-bladder. The food should be plain and readily digestible. Saccharin matters, fat meats, and highly seasoned dishes should be avoided. Water-drinking between meals should be encouraged. Regular exercise in the open air, provided the symptoms are latent, is extremely beneficial.

Digestive disturbances should receive appropriate treatment. Among drugs, alkalis and alkaline mineral waters are undoubtedly efficacious. Sodium bicarbonate or sodium phosphate may be taken well diluted in the morning an hour before breakfast and also between meals. If there is decided constipation, a small quantity of Rochelle salt or sodium sulphate may be added to each potation. The natural mineral waters, notably those of Carlsbad and Vichy, have acquired a high reputation. When there is a tendency to so-called bilious attacks, an occasional course of calomel in fractional doses will be found of benefit.

Surgical intervention is called for: (1) When, despite medical treatment, attacks of colic occur so frequently and are of such severity as to cause disability or make the addiction to morphin a likelihood; (2) in persistent obstruction of the common duct; (3) in hydrops of the gall-bladder due to impaction or stricture of the cystic duct; and (4) in suppurative inflammation of the gall-bladder or gall-ducts.

Hepatic Colic.—Morphin (¼ grain) and atropin ($\frac{1}{150}$ grain) should be given hypodermically. Agonizing pain often yields very promptly to a few whiffs of chloroform. In the mild but rather persistent attacks a few doses of antipyrin in hot water may suffice. The external application of heat (poultice or hot bath) is very useful.

When vomiting is urgent, carbonated water or champagne may be given. In threatened collapse diffusible stimulants are needed.

Obstruction of the Common Duct.—The measures best suited for promoting the advance of the stone into the bowel are rest, regulation of diet, the free use of alkaline mineral waters, the occasional exhibition of saline laxatives, and the application of heat to the hypochondriac region. Olive oil has been recommended as a special remedy, but it is of doubtful efficacy. As the sequelæ of impaction of the common duct are so grave, surgical aid should be invoked if the obstruction is not removed under medical treatment within a period of three or four weeks.

HYPEREMIA OF THE LIVER. 11.9.

Varieties.—(1) Active hyperemia. (2) Passive hyperemia.

Etiology.—*Active hyperemia* is commonly due to dietetic indiscretions. It may result from overindulgence in alcohol. It is often present in the infectious fevers. It appears to arise idiopathically in hot climates.

Passive hyperemia results from diseases that obstruct the venous circulation, as chronic heart and lung disease.

Pathology.—The liver is enlarged and filled with blood. In the *passive* variety, the center of the lobule, the area of the hepatic vein, is deeply pigmented, while the periphery, the area of the portal vein, is pale. This mottled appearance has given rise to the term "nutmeg liver." In persistent cases pigmentation, atrophy of the liver-cells, and overgrowth of the connective tissue result—a condition termed "cyanotic induration."

Symptoms.—*Active Hyperemia.*—The liver is enlarged and somewhat tender. There is a sense of fulness or even actual pain in the hepatic region. There may be slight jaundice. Digestive disturbances—anorexia, nausea, flatulence, headache, and epigastric tenderness usually coexist.

In the *passive variety* the symptoms are much the same, though less marked. The liver is often quite large, and in extreme cases, such as follow tricuspid regurgitation, it may pulsate.

Prognosis.—In simple active congestion the prognosis is good. In passive congestion the prognosis depends on the cause.

Treatment.—Active hyperemia from dietetic errors usually yields promptly to restriction of the diet and the administration of a mercurial purge, followed by a saline—Rochelle salt, Seidlitz powder, or sodium phosphate. In recurring attacks, in addition to hygienic and dietetic regulations, a pill like the following often proves useful:

℞. Massæ hydrargyri . . . . gr. v
Pulveris rhei
Extracti gentianæ . . . . āā ʒss
Olei caryophylli . . . . gtt. iv.—M.
Fiant pilulæ No. xx.
SIG.—One or two occasionally, as directed; to be continued, if required, thrice daily for several days.

In passive congestion treatment must be directed to the primary disease. In mild cases alkaline mineral waters (Carlsbad, Congress, and Friederichshall) do well. A mercurial laxative may be used from time to time. In severe cases the most effective measures are absolute rest, a milk diet, saline purges, and wet-cupping over the liver.

CIRRHOSIS OF THE LIVER.

(Chronic Interstitial Hepatitis.)

Definition.—A chronic disease of the liver characterized by a hyperplasia of the connective tissue and more or less extensive retrograde changes in the liver-cells.

Varieties.—The most important varieties are *atrophic cirrhosis*, *hypertrophic cirrhosis*, *syphilitic cirrhosis*, *biliary cirrhosis*, and *capsular cirrhosis.*

ATROPHIC CIRRHOSIS.

(Laennec's Cirrhosis; Alcoholic Cirrhosis: Gin-drinker's Liver.)

Etiology.—It occurs most commonly in males of middle age. The chief cause is the continued use of alcohol, especially in the form of raw spirits. Syphilis may also cause this type of cirrhosis. It is possible that some cases owe their origin to the specific fevers.

Pathology.—In the earliest stages the liver is somewhat enlarged from hyperemia, but when the process is advanced the organ is small, hard, and covered with numerous small nodules or granulations ("hob-nails"). A section of the liver reveals a network of fine and coarse pearly bands of connective tissue. The contraction of this connective tissue is responsible for the reduction in the size of the organ and the granular surface.

Microscopic examination reveals an overgrowth of connective tissue of a fibrous or cicatricial character, and chiefly interlobular in distribution. The shrinking of this tissue compresses the portal veins and causes degeneration and atrophy of the liver-cells.

Symptoms.—Obstruction of the portal circulation first causes *congestion and catarrh of the stomach*, hence the initial symptoms are anorexia, fetor of the breath, fulness and distress after eating, eructations, nausea, vomiting of mucus, flatulence, and constipation. For months and even years these phenomena may be the only evidence of the disease. As the pressure in the portal system increases, the collateral vessels enlarge, and as a result the *superficial abdominal veins become prominent* and *hemorrhoids* develop. Engorgement of the portal system also leads to *ascites* and swelling of the feet, to *enlargement of the spleen*, and, not infrequently, to copious *hemorrhage* from the stomach or bowel.

At first the liver is somewhat enlarged; later, however, the area of percussion-dulness is distinctly reduced. There is a gradual loss of flesh and strength. The skin is muddy in appearance, but conspicuous jaundice is very uncommon. Nervous symptoms—delirium, stupor, convulsions, and coma—occasionally appear toward the end of the disease. They are probably due to the retention of poisons that the liver is unable to convert or to eliminate.

The majority of cases terminate fatally in from three to five years, or in from one to two years after the compensatory circulation fails. Death results from exhaustion, hemorrhage, pulmonary edema, intercurrent disease, or toxemia.

Complications.—The kidneys, heart, and blood-vessels are often coincidentally involved in the cirrhotic process. Tuberculosis, especially of the peritoneum, is a very common complication.

Diagnosis.—In the early stage the diagnosis can only be suspected. In the drunkard, chronic gastric catarrh with enlargement of the liver would strongly indicate the disease.

Thrombosis of the portal vein produces the same clinical picture, but the symptoms usually develop much more rapidly.

Chronic Peritonitis with Effusion.—This is usually tuberculous or cancerous. The history, abdominal tenderness, the detection of localized masses or ill-defined indurations, the presence of other foci of disease, the high specific gravity (above 1014) of the ascitic fluid, and the absence of symptoms indicating portal obstruction will generally suggest chronic peritonitis.

Prognosis.—The outlook for permanent relief is bad.

Treatment.—Alcohol must be interdicted. A diet of bland, readily digested food is indicated. The gastric catarrh should receive appropriate treatment. Lavage of the stomach is contraindicated on account of the presence of esophageal varicosities. Potassium iodid is of service in syphilitic cases, but not otherwise. Ammonium chlorid (10 grains three times a day) is sometimes useful. Portal congestion is best relieved by the administration of salines (sodium phosphate or Rochelle salt) in hot water one-half hour before breakfast.

Ascites can sometimes be removed by the administration of cathartics and diuretics. A concentrated solution of Epsom salts (½ to 1 ounce), taken in the morning before breakfast, is usually the most efficient purgative. Occasionally it may be desirable to substitute compound jalap powder or elaterium. The diuretics of approved value are potassium acetate or bitartrate, digitalis, and squills.

Niemeyer's pill has a well-deserved reputation:

℞. Massæ hydrargyri
Pulveris digitalis
Pulveris scillæ, ... āā gr. xx.—M.
Fiant pilulæ No. xx.
SIG.—One pill thrice daily.

When the ascites is large and does not yield readily to drugs, paracentesis should be practised (see p. 116).

Surgical Treatment.—Talma's operation (suture of the omentum to the margin of the abdominal incision and irritation of the peritoneal surfaces of the liver) or one of its modifications, has proved of some benefit in a limited number of cases of liver cirrhosis with ascites. The object of the operation is to establish a compensatory circulation by making accidental adhesions and thus increasing the anastomoses between the vessels of the portal system and those of the systemic circulation. The operation is contraindicated when cardiac or renal disease coexists.

HYPERTROPHIC CIRRHOSIS.

(Hypertrophic Cirrhosis of Hanot.)

Pathology.—The causes of hypertrophic cirrhosis are not understood. Alcohol does not appear to be a factor. It is seen chiefly in men between twenty and thirty years of age. The liver is greatly enlarged throughout the entire course of the disease. The organ is of yellowish or greenish color, and its surface is smooth or finely granular.

Microscopically, a proliferation of connective tissue is found, but the latter is chiefly *intralobular*, is more cellular than fibrous, and shows little tendency to contract. The liver-cells remain intact and not infrequently share in the proliferation.

Symptoms.—The liver is much enlarged permanently, often tender, and the seat of recurrent attacks of pain. Jaundice of a mild type is rarely absent. The stools, however, retain their normal color. The spleen is enlarged. Hemorrhages into the skin and from mucous membranes are not uncommon. Toward the end of the disease, symptoms of hepatic intoxication may develop. Ascites, profuse hematemesis, and enlargement of the superficial abdominal veins are rarely observed. The course is long—often from five to ten years.

The **treatment** is that of congestion of the liver. Calomel and potassium iodid have been recommended.

OTHER FORMS OF CIRRHOSIS OF THE LIVER.

Syphilitic Cirrhosis of the Liver.—In the diffuse form the appearance of the liver is similar to that of alcoholic cirrhosis. In the gummatous form, however, the organ is enlarged and often coarsely lobulated from fibrous transformation of the gummata. A history of syphilitic infection, an enlargement of the liver that is grossly nodular, and a fair preservation of the general health will suggest the condition. The disease often responds favorably to antisyphilitic treatment.

Biliary Cirrhosis of the Liver.—This form is due to stasis of the bile and results from persistent obstruction of the bile-ducts by calculi, stricture, or tumor. Clinically, it resembles the hypertrophic cirrhosis of Hanot, but, unlike the latter, the jaundice is very intense and develops rapidly, the stools are devoid of bile, the liver is only moderately enlarged, and the course is short—rarely more than two or three years.

Capsular Cirrhosis (Chronic Perihepatitis).—This form is characterized by enormous thickening of the capsule of the liver. The symptoms closely resemble those of atrophic cirrhosis, but the course is extremely slow, the ascites returning again and again after tapping. In many cases interstitial nephritis, chronic capsulitis of the spleen, chronic peritonitis, and pericarditis are also present.

ABSCESS OF THE LIVER.

(Acute Suppurative Hepatitis.)

Etiology.—Abscess of the liver is probably always due to the action of micro-organisms—Amœba coli, streptococcus, staphylococcus, colon bacillus. They may enter the liver through the portal vein, hepatic artery, or bile-ducts.

(1) Amebic dysentery is a very common cause, the amebæ entering the liver through the portal vein. Occasionally amebic abscesses occur without any evidence of dysentery. (2) Septic emboli from gastric ulcers, duodenal ulcers, purulent appendicitis, etc., may also lodge in the branches of the portal vein and thus excite a suppurative inflammation (suppurative pylephlebitis). (3) Pyogenic organisms may enter the hepatic artery in ulcerative endocarditis, abscess of the lungs, and general pyemia. (4) Suppuration by way of the bile-ducts sometimes occurs in angiocholitis secondary to gall-stones. (5) Traumatism may be a causal factor. (6) Finally, suppuration may result from the secondary infection of an echinococcus cyst.

Pathology.—The abscesses following amebic dysentery ("tropical abscess") and traumatisms are generally solitary, and usually occupy the right lobe. Metastatic abscesses are multiple.

Events.—Hepatic abscess may kill by septic poisoning or by perforation into the lung, abdomen, pleura, pericardium, or vena cava. Recovery may follow operation or spontaneous rupture into the bronchi, into the stomach or bowel, or externally.

Symptoms.—**Local Symptoms.**—The liver is enlarged and tender. The enlargement is more often upward than downward. Circumscribed bulging beneath the costal arch is sometimes noted. Fluctuation is occasionally detected. There is usually severe pain in the liver region and right shoulder. Exploratory puncture may reveal pus. Slight jaundice may develop, but it is often absent.

Constitutional Symptoms.—These result from sepsis, and include fever of a remittent or irregular type, chills, profuse sweating, marked anemia, and leukocytosis.

Rupture into the lung is characterised by severe cough, weak breathing at the base of the right lung, and the expectoration of brownish matter, sometimes containing amebæ.

Diagnosis.—**Hydatid cysts** develop slowly, are not painful, are not associated with septic phenomena, and yield clear fluid on aspiration.

Cancer of the Liver.—The history, marked cachexia, involvement of other organs, presence of jaundice, detection of hard nodules on the surface of the liver, and the absence of septic phenomena will suggest cancer.

Intermittent Fever due to Impacted Gall-stones.—In this condition the pain, fever, and sweating are often periodic; the health in the intervals may be well preserved; the jaundice increases at each paroxysm; the symptoms may persist for several years.

Prognosis.—Embolic abscesses are invariably fatal. Traumatic and amebic abscesses may terminate favorably upon spontaneous or induced evacuation.

Treatment.—In multiple abscesses treatment is palliative. Large solitary abscesses should be opened and drained.

CANCER OF THE LIVER.

Etiology.—Cancer of the liver is more common in men than in women. It is infrequent before the age of forty. Heredity, traumatism, and chronic irritation from gall-stones are predisposing factors.

Pathology.—Primary cancer of the liver is rare; secondary cancer is common. The primary form may appear as a single large nodule (*massive cancer*) or as a wide-spread infiltrating growth (*nodular cancer*). The latter form is sometimes associated with cirrhosis of the liver (*cirrhotic cancer*). The secondary variety is usually due to the lodgment in the portal capillaries of cancerous emboli derived from a primary growth in one of the neighboring organs, especially the stomach. The liver is much enlarged, and studded with numerous grayish-white nodes, some of which project from the surface. The superficial nodes are often depressed at the center.

Symptoms.—(1) The liver is enlarged and very painful, and often presents one or more smooth, hard nodules. The latter may show a central depression. (2) Cachexia is pronounced and develops rapidly. (3) Jaundice is common, but it is rarely intense. (4) Digestive disturbances are a prominent feature, and often precede the hepatic symptoms. Ascites sometimes results from portal obstruction. Toward the end, slight fever, delirium, stupor, and coma may develop (hepatic intoxication).

Diagnosis.—**Hypertrophic cirrhosis** may be distinguished by the smooth, uniform enlargement of the liver, the enlargement of the spleen, the persistence of icterus without loss of color in the stools, the absence of marked cachexia, the age of the patient (between twenty and forty), and the slow course.

Abscess.—This may be distinguished by the history, the septic fever, and the results of exploratory puncture.

Syphilis of the Liver.—The history of specific disease, the age, and the absence of cachexia will aid in the diagnosis.

Prognosis and Treatment.—Absolutely unfavorable. The duration is from a few months to a year. Treatment can only be palliative.

HYDATID CYST OF THE LIVER.

See Osler 32

(Echinococcus of the Liver.)

Etiology and Pathology.—Hydatid cysts are formed by the embryos of the Tænia echinococcus, a small tapeworm inhabiting the intestines of the dog. The disease is common in Iceland, Australia, and some parts of Europe, but is rare in America.

The eggs of the worm are accidentally ingested by man, and embryos are liberated in the stomach, whence they may migrate to any organ; the liver, however, is most commonly affected through the portal vein. The fixed embryo soon develops into a cyst that is composed of an external laminated layer and an internal breeding layer. A connective-tissue layer is formed on the outside from irritation.

The cyst contains a clear, non-albuminous fluid that has a specific gravity of 1005 to 1007, and which is rich in chlorids.

Scolices or larvæ develop from the breeding layer; they are provided with four suckers and a circle of hooklets, and produce daughter-cysts within the parent-cyst. When ingested by the dog, the larvæ develop into mature tapeworms.

Symptoms.—Small cysts excite no symptoms. Large cysts produce an irregular enlargement of the liver, with a sense of weight or fulness in the hypochondriac region. If the cyst is superficial, an elastic, fluctuating tumor may be detected on palpation. On percussion a peculiar vibratory sensation (hydatid thrill) may be imparted to the hand. Aspiration yields a clear fluid containing the characteristic hooklets. Fever, pain, and jaundice are usually absent.

Events.—(1) The cyst may reach a certain size and then become quiescent. (2) Trifling injury may convert it into abscess. (3) Rupture of the cyst into neighboring organs may terminate in death or in recovery.

Diagnosis.—The diagnostic features are a smooth, tense, elastic tumor of the liver, of slow growth, without pain, fever, or pronounced disturbance of the general health, and yielding, upon exploratory puncture, a clear fluid containing hooklets.

Prognosis.—In uncomplicated cases the prognosis is guardedly favorable.

Treatment.—Aspiration under antiseptic precautions is sometimes followed by permanent recovery. Surgeons of the largest experience, however, prefer free incision and evacuation of the cysts. Purulent cysts should be treated as abscesses.

AMYLOID LIVER.

(**Waxy Liver; Lardaceous Liver.**)

Definition.—An enlargement of the liver due to the deposition of a peculiar albuminoid substance.

Etiology.—The chief causes are prolonged suppuration, especially of bones; tuberculosis; syphilis; and long-standing cachexia.

Pathology.—The liver is uniformly enlarged, hard, and smooth. The margins are blunt. On section, the surface presents a translucent, wax-like appearance, and is colored mahogany brown with Lugol's solution. The degenerative process begins in the walls of the blood-vessels and spreads to the connective tissue.

Symptoms.—The liver is uniformly enlarged, smooth, firm, and painless, and presents a rounded edge. The spleen and kidneys almost always share in the degeneration, and, in consequence, the spleen is enlarged and hard and the urine contains albumin and tube-casts. Anemia and emaciation are often pronounced. Jaundice and ascites are uncommon.

Diagnosis.—This is based on the history, the uniform enlargement of the liver, the absence of pain, of jaundice, and of ascites, and the involvement of other organs. In **leukemia** the liver and spleen are often uniformly enlarged, but an examination of the blood will prevent an error in diagnosis.

Prognosis and Treatment.—The prognosis depends somewhat upon the curability of the primary disease. The outlook, however, is always grave. The treatment must be directed to the causal disease.

ACUTE YELLOW ATROPHY OF THE LIVER.

(**Acute Parenchymatous Hepatitis; Malignant Jaundice.**)

Definition.—A very rare and grave disease characterized anatomically by a rapid destruction of the liver tissue, and manifested clinically by jaundice, hemorrhages, a reduction in the size of the liver, and marked cerebral phenomena.

Etiology.—The disease occurs more frequently in women than in men. It is usually seen between the ages of twenty and thirty. Pregnancy is a predisposing factor. Alcoholic excesses, syphilis, and emotional excitement have been given as exciting causes. The rapid course, widespread lesions, and the fact that the disease has occurred endemically suggest a toxic or infectious origin.

Pathology.—The liver is reduced in size, flaccid, and friable. The surface is yellowish red and mottled. Microscopic examination reveals advanced necrosis of the liver-cells, hemorrhagic extravasations, hematogenous pigmentation, and occaasionlly small-celled infiltration. The other organs are usually the seat of fatty and parenchymatous degeneration.

Symptoms.—(1) The initial symptoms are those of catarrhal *jaundice*. (2) Nervous symptoms (*cholemia*) soon follow; these are severe headache, maniacal delirium, stupor, and coma. (3) The urine is scanty, and usually contains *leucin* and *tyrosin*, bile, albumin, and tube-casts. The excretion of urea is often greatly diminished. (4) The area of hepatic percussion dulness rapidly decreases. (5) Hemorrhages from the mucous membranes and into the skin are common. Fever is usually absent. The disease rarely lasts longer than a week or ten days. Recovery is extremely rare.

Diagnosis.—In *acute phosphorus-poisoning* acute gastritis precedes the jaundice, the vomitus and stools may be phosphorescent or have the odor of phosphorus, the liver is generally enlarged, and the urine contains much sarcolactic acid.

In *hypertrophic cirrhosis* the liver is enlarged and often painful, the course is slow, and leucin and tyrosin rarely appear in the urine.

Treatment.—This must be symptomatic.

ACUTE PERITONITIS.

Definition.—An acute inflammation of the peritoneum. The process may be general or localized.

Etiology.—The disease is probably always caused by bacteria, which enter the peritoneum from the neighboring viscera, especially the alimentary canal, from the Fallopian tubes, from external wounds, or directly from the blood. The organisms most frequently found are the Streptococcus pyogenes, Staphylococcus pyogenes, Bacillus coli, pneumococcus, Bacillus pyocyaneus, and gonococcus.

Peritonitis may follow—(1) *Perforation* of the peritoneum by an external wound, by rupture of a gastric or intestinal ulcer, by rupture of a suppurating appendix, gall-bladder, or Fallopian tube, or by rupture of a visceral abscess; (2) *extension* of a septic process in adjacent structures—stomach, bowel, gall-bladder, pancreas, uterus; (3) *traumatism;* (4) *general infections*—septicemia, specific fevers, rheumatism, tuberculosis, etc.

Pathology.—The serous surfaces first become red and lusterless; later a serofibrinous, fibrinous, or purulent exudate is formed. Putrid and hemorrhagic exudates are sometimes observed.

Symptoms.—The most prominent symptoms are intense abdominal pain and tenderness. The breathing is shallow and thoracic. To relax the abdominal parietes, the patient lies motionless upon his back, with the legs and thighs flexed. The features are pinched, and the expression is anxious. The abdomen is distended, and its walls are rigid. Percussion at first reveals general tympany, but later there may be dulness in the flanks from the gravitation of the exudate. The temperature is usually moderately high (102°–104° F.), and the pulse is small, rapid, and "wiry." The bowels are usually constipated. Vomiting and hiccup are common symptoms. In severe cases collapse speedily ensues, and is indicated by a fall in the temperature, a cold, clammy surface, a rapid, feeble pulse, and suppression of urine.

In *localized* peritonitis the constitutional symptoms are less severe. Pain, tenderness, and rigidity are circumscribed. General tympanites is usually absent. Abscess formation is common.

Diagnosis.—**Acute Enteritis.**—In this disease the pain is colicky and less intense; tenderness is much less marked; rigidity is rarely present; there is diarrhea; the constitutional symptoms are not so grave.

Intestinal Obstruction.—Constipation is absolute; vomiting is stercoraceous; fever and abdominal tenderness are less pronounced.

Hysteric Abdomen.—This condition may resemble peritonitis in all particulars. The personal history must be carefully considered. Fever is not usually present, the pulse is not usually rapid and wiry, and when the attention is distracted, the pain may disappear.

Prognosis.—Diffuse septic peritonitis is almost invariably fatal. The duration is usually from two to six days. Life is occasionally saved in perforative peritonitis by prompt operation. In localized peritonitis the outlook is much more favorable.

Treatment.—Early operation offers the only hope of saving life in perforative or septic cases. Apart from laparotomy, treatment is, for the most part, palliative. When the stomach is retentive, small quantities of milk and lime-water or of broth may be given by the mouth. Ice may be given to suck. If vomiting be persistent, nutrient enemas are to be given. Locally, either very cold or very hot applications may afford relief. Opium is useful in allaying pain, controlling vomiting, and diminishing peristaltic movements. Remarkably large doses are often well borne. In non-perforative cases saline purgatives in concentrated solution (1 to 2 drams every two hours) may be given until the bowels move freely. These salts, while not increasing peristalsis, attract serum from the turgid blood-vessels and thus relieve congestion.

CHRONIC DIFFUSE PERITONITIS.

Etiology.—Chronic peritonitis may be a sequel of acute peritonitis. In a few instances it has seemed to have resulted from syphilis. In the vast majority of cases it is tuberculous or cancerous.

Pathology.—The intestines are matted together by bands of fibrous lymph. The omentum is often contracted and greatly thickened. Effusion is usually present, but it varies considerably in amount; it is highly albuminous, and in the tuberculous and cancerous varieties it may be bloody.

Symptoms.—Fever is slight and may be absent. Pain is not severe, and is frequently paroxysmal. There is usually more or less diffuse tenderness. Anemia and emaciation are often pronounced. The abdomen is generally distended; often irregularly, from sacculated effusions, inflated intestinal coils, or the projecting matted omentum. Palpation often detects a friction fremitus and resistant masses or nodules. Percussion yields dulness, varying in extent with the amount of effusion. When the fluid is sacculated, the dulness may be irregularly distributed. Fluctuation can sometimes be elicited. On tapping, the fluid is turbid, rich in albumin, and of high specific gravity (about 1015). In cancerous and tuberculous cases it is frequently bloody.

Diagnosis.—The diagnosis between *tuberculous* and *cancerous* peritonitis is not always easy. The tuberculous form usually occurs in persons under forty, gives rise to less cachexia than the cancerous form, and is frequently associated with tuberculous foci elsewhere, especially in the lung, pleura, testis, or Fallopian tube. In doubtful cases the tuberculin test may be employed or a guinea-pig may be inoculated with the exudate.

Prognosis.—Cancerous peritonitis is invariably fatal. Tuberculous peritonitis, while always grave, not infrequently ends in recovery, especially in children.

Treatment.—In the tuberculous form the general treatment should be that of pulmonary tuberculosis. When the effusion is large, aspiration will be required. Surgical treatment (free incision with washing-out of the abdominal cavity with normal salt solution) should be advised in suitable cases.

ASCITES.

Definition.—A collection of serous fluid in the peritoneal cavity.

Etiology.—(1) It may result from the causes of general dropsy—heart disease, nephritis, chronic lung disease, and anemia. (2) It may be due to obstruction of the portal circulation, from cirrhosis of the liver, tumor, or thrombosis of the portal vein. (3) It occurs in chronic peritonitis. (4) It is often caused by tumors of the abdomen.

Symptoms.—When the effusion is large, a sensation of weight in the abdomen, dyspnea, scanty urination, and edema of the feet may result from pressure.

Physical Signs.—**Inspection.**—The abdomen is distended; the surface is smooth and shining; the base of the thorax is broadened; the navel is more or less obliterated; the superficial veins are frequently enlarged; and, when the patient lies in the dorsal position, the flanks bulge.

Palpation may elicit fluctuation, and in the flanks a sense of resistance.

Percussion reveals dulness and resistance in dependent parts, with superincumbent tympany. The dulness is moveable and is detected in the flanks when the patient occupies the dorsal position.

Aspiration.—The fluid is usually clear, straw-colored, and albuminous. The specific gravity is from 1010 to 1020. In cancerous and tuberculous peritonitis the fluid is sometimes bloody. Occasionally, chylous fluid is present.

Diagnosis.—**Tympanites.**—This yields universal hyperresonance on percussion.

Ovarian Cysts.—The enlargement is at first unilateral. As the intestines are pushed aside, the dulness is anterior and the resonance is in the flanks. Vaginal examination often furnishes important data. The fluid of the cyst has a higher specific gravity (1025).

Distention of the Bladder.—The history, the location of the dulness, and the results of catheterization will render the diagnosis apparent.

Treatment.—Treatment should be directed to the original cause. Hydragogue cathartics and diuretics are sometimes useful. Concentrated saline solutions, compound jalap powder (20 to 40 grains), and elaterium (⅛ grain) are the most useful cathartics. Infusion of digitalis (3 to 4 fluidrams), citrated caffein (2 to 3 grains), potassium citrate or acetate (20 grains), and Niemeyer's pill (see p. 105) are the most reliable diuretics.

℞. Potassii citratis . . . . ℥ss
Infusi digitalis . . . . f℥vj.—M.
SIG.—A tablespoonful thrice daily.

When the effusion is large and causes discomfort or great diminution in the quantity of urine, paracentesis should be performed.

Paracentesis Abdominis.—The bladder having been emptied, the patient is placed in a semirecumbent position, and a spot in the median line midway between the umbilicus and the symphysis pubis is anesthetized by means of a block of ice sprinkled with salt. A stout trocar is now introduced with a quick thrust into the abdominal cavity, a rubber tube is attached to the cannula for the purpose of conveying the fluid into a pail placed below the patient's bed, and the trocar is then withdrawn. While the fluid is escaping, a many-tailed bandage is adjusted to the abdomen and gradually tightened. The application of such a binder should never be omitted. It gives support to the relaxed abdominal walls, and tends to prevent syncope and hematemesis. When the fluid ceases to flow, the cannula is removed, and the opening sealed with an antiseptic pad and a few strips of adhesive plaster.

In tuberculosis, peritonitis, and in cirrhosis of the liver with recurrent ascites surgical treatment sometimes proves successful.

DISEASES OF THE KIDNEYS.

THE URINE.

Normal urine is a pale, amber-colored fluid, of acid reaction, having a specific gravity of 1015 to 1025, and amounting in quantity to about 50 ounces (1500 c.c.) in twenty-four hours.

Polyuria.—An increased flow of urine.

Temporary polyuria may result from—(1) Excessive ingestion of fluids. (2) Administration of diuretics. (3) Suppression of perspiration. (4) Crises of certain febrile diseases, and certain neurotic manifestations, such as neuralgia and hysteria. (5) Absorption of serous effusions and transudations. (6) Removal of some temporary obstruction in the urinary passages.

Permanent polyuria may result from—(1) Diabetes mellitus. (2) Diabetes insipidus. (3) Chronic interstitial nephritis. (4) Amyloid kidney.

Diminution of the amount of urine or suppression of urine (anuria) occurs—(1) When there is excessive secretion through other channels, as in perspiration and diarrhea; (2) in fever; (3) in severe congestion of the kidneys; (4) in acute nephritis and late in chronic parenchymatous nephritis; (5) in collapse; (6) in certain nervous conditions, as in some cases of hysteria; and (7) from mechanical obstruction, as in compression of the ureters by tumors and in enlargement of the prostate gland.

Urea.—Urea is the final product of the decomposition of proteids in the body. A large part is formed in the liver. It is completely soluble in urine, but the nitrate of urea crystallizes in the form of transparent imbricated plates when nitric acid is added to urine that has been partially evaporated.

The amount of urea excreted varies greatly in health. Normal urine contains about 2 per cent. of urea. The average daily amount excreted is from 300 to 600 grains.

The excretion of urea is *increased*—(1) After the ingestion of much albuminous food; (2) after exertion; (3) in febrile and acute inflammatory diseases; (4) in diabetes; (5) after the use of certain drugs, as thyroid extract, caffein, and salicylic acid.

The excretion of urea is *diminished*—(1) In nephritis; (2) in inanition and cachexia; (3) in destructive diseases of the liver—acute yellow atrophy, cirrhosis.

Fowler's Hypochlorite Test for Urea.—Add to 1 volume of the urine 7 volumes of Labarraque's solution of chlorinated soda. Shake the jar containing the mixture occasionally, and stand it aside for two hours, when the urea will have been decomposed. Now take the specific gravity of the quiescent fluid.

Ascertain the specific gravity of the mixture of urine and Labarraque's solution before decomposition. To do this, multiply the specific gravity of the pure Labarraque's solution by 7, add this to the specific gravity of the pure urine, and divide by 8. The result is the specific gravity of the mixed fluid. From this subtract the specific gravity of the quiescent mixture after decomposition of the urea, multiply the difference by 77, and the result is the percentage of urea (Tyson).

Lithuria.—Uric acid or urates in the urine. These substances appear to be derived from the nuclein of cellular nuclei. When they are in excess, the urine is heavy, dark in color, and on cooling throws down a brick-red deposit.

Microscopically, uric acid appears as reddish-yellow rhombic prisms or lozenge-shaped crystals.

Amorphous urates appear as fine, dark, and opaque granules.

Crystalline urates appear as needles, dumb-bells, or as globular masses from which sharp spines project.

Murexid Test for Uric Acid and its Salts.—Evaporate a little urine in a porcelain dish, add a drop or two of strong nitric acid, and heat again to dryness. Cool, and add a drop of liquor ammoniæ, and the beautiful purple color of murexid is developed.

Urates.—The urates are present in small quantity in normal urine. They may become perceptible or transiently increased: (1) In urine exposed to a cold atmosphere. (2) In urine made scanty by free perspiration or diarrhea. (3) When the acidity of the urine is temporarily increased. (4) After excessive indulgence in nitrogenous food.

The urates are increased pathologically in many diseases which directly or indirectly interfere with tissue or food metabolism, notably in: (1) Gout; (2) fever; (3) leukemia; (4) indigestion; (5) diseases of the lungs—from interference with oxidation.

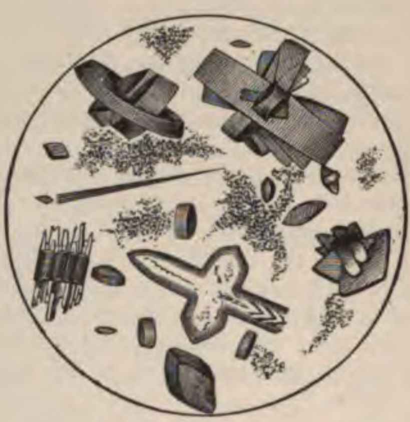

FIG. 3.—Uric acid and uric acid salts.

Leucinuria and Tyrosinuria.—Leucin and tyrosin are found in the urine in certain specific fevers, and especially in fatty degeneration of the liver resulting either from phosphorus-poisoning or from acute yellow atrophy.

They may be detected by evaporating a few drops of the urine on a glass slide. Leucin appears in the form of small, round, glistening spheres, resembling fat-drops, but, unlike the latter, they are insoluble in ether. Tyrosin appears in the form of intersecting tufts of fine acicular crystals.

Phosphaturia.—Phosphates occur in two forms, amorphous and crystalline.

Amorphous earthy phosphates are found in alkaline urine, and are precipitated by adding a few drops of liquor ammoniæ to the urine.

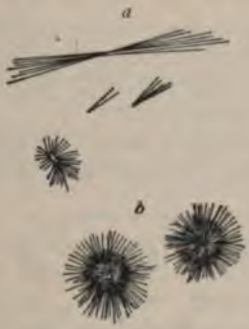

FIG. 4.—*a*, Tyrosin crystals; *b*, leucin crystals.

Crystallized phosphate of lime appears as stellar or rod-shaped crystals which are soluble in acetic acid.

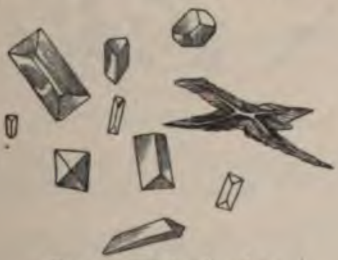

FIG. 5.—Triple phosphate.

The ammoniomagnesium phosphate, or triple phosphate, appears in decomposing urine as transparent, coffin-shaped prisms. They may resemble crystals of oxalate of lime, but, unlike the latter, are freely soluble in acetic acid.

The presence of phosphates in the urine is no indication of excess, for when normal in amount, they are often precipitated in urine that is temporarily alkaline.

The detection of triple phosphates in newly voided urine indicates decomposition in the bladder, a condition resulting from vesical catarrh.

Phosphaturia results from many causes—certain nervous diseases, nervous dyspepsia, rickets, osteomalacia, leukemia, and gout. Cases of polyuria with phosphaturia have been described (*diabetes phosphaticus*).

Chlorids.—The quantity of these salts is *increased:* (1) After exertion. (2) During the absorption of serous effusions.

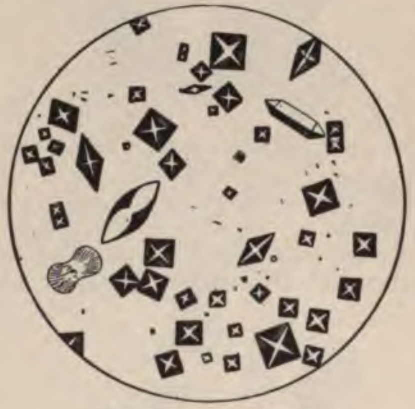

FIG. 6.—Oxalate of lime.

The quantity is *decreased:* (1) In most febrile diseases. (2) In nephritis. (3) In many wasting diseases. (4) Especially in pneumonia.

Test.—We may thus roughly estimate the quantity. Add a few drops of strong nitric acid to the urine, remove any albumin that may be present, and then add to the clear urine a little of a strong solution of nitrate of silver. The abundance of the white precipitate will indicate the quantity of chlorids present.

Oxaluria.—Oxalate of lime appears in the urine as dumb-bell-shaped crystals or as minute, highly refracting octahedra.

They are found in excess: (1) After eating certain fruits and vegetables, as spinach, rhubarb, cauliflower, and pears; (2) in certain nervous diseases, notably hypochondriasis, melancholia, and neurasthenia; (3) in diabetes; (4) various digestive disturbances.

Tube-casts.—These are cylinders of albuminoid substances formed in the uriniferous tubules. They are often composed in part of epithelial cells, blood-cells, or the products of degenerated cells. They appear as:

Hyaline Casts.—These are clear, translucent cylinders, often so pale as to be scarcely visible. They occur in the urine in all forms of nephritis, in congestion of the kidneys, in jaundice, and even in health. They are frequently the only casts present in chronic interstitial nephritis.

Waxy Casts.—These resemble hyaline casts, but they appear more solid and rigid and are more or less yellow. They occur especially in chronic parenchymatous nephritis.

Epithelial Casts.—These are cylinders of epithelial cells or hyaline casts covered with epithelial cells. They occur especially in acute parenchymatous nephritis.

Granular Casts.—These are cylinders covered with the débris of broken-down epithelial cells. They may occur in any form of nephritis.

Fatty Casts.—These are casts studded with oil-drops derived from degenerated epithelium. They occur chiefly in chronic parenchymatous nephritis.

Blood Casts.—These are cylindric masses of red blood-cells, or, more commonly, hyaline casts studded with red blood-cells. They occur in acute and chronic hemorrhagic nephritis.

Pus and Bacterial Casts.—Casts composed respectively of masses of pus-cells and of bacteria are occasionally met with in suppurative nephritis.

Cylindroids.—These formations may resemble hyaline casts, but they are usually much longer and often taper off at one end to a thread. Moreover, they frequently show constrictions at different points. Their presence is not a proof of nephritis. They often occur in conditions of renal irritation.

Urobilinuria.—Urobilin is probably a derivative of bilirubin. When present in excess, the urine is dark brown. When deposited in the tissues, it causes a brownish pigmentation known as urobilin-icterus. The urobilin in the urine may be pathologically increased—(1) In diseases associated with destruction of red blood-cells, as pernicious anemia and scurvy; (2) after the absorption of hemorrhagic effusions; (3) in acute infectious diseases; (4) in certain liver diseases (cancer, cirrhosis, catarrhal jaundice), provided the entrance of bile into the bowel is not entirely prevented.

Hematoporphyrinuria.—Hematoporphyrin is a product of the decomposition of hemoglobin. It is hematin deprived of its iron. Large amounts impart to the urine a dark-red color. It is found in the urine in a large number of diseases, and in chronic poisoning by sulphonal and trional.

Glycosuria.—Grape-sugar in the urine.

Causes.—Normal urine contains a trace, but this is not recognizable by the ordinary tests. Decided glycosuria is seen—(1) In diabetes mellitus; (2) after the digestion of large amounts of saccharine matter; (3) in poisoning by certain drugs, such as phloridzin, nitrites, chloroform; (4) in pregnancy; (5) in diseases or injuries to the floor of the fourth ventricle; (6) in lesions of the pancreas involving the islands of Langerhans; (7) in many nervous diseases and acute infections—epilepsy, tetanus, cholera, pertussis, etc.

Qualitative Tests for Glucose.—The copper tests are commonly employed, and depend on the power which glucose possesses of converting blue oxid of copper into the orange-yellow suboxid.

Trommer's Test.—Add to the suspected urine half its volume of liquor potassæ, and if any precipitate falls, filter the solution; then add one or two drops of a weak solution (1 : 30) of sulphate of copper, and heat the resulting mixture. If sugar is present, a dense yellow or red precipitate falls.

Simple decolorization of the fluid is no proof of sugar.

Fehling's Test.—As the fluid employed in this test spoils on keeping, it should be freshly prepared when required by mixing in equal proportions the following solutions:

First solution: Dissolve 34.64 grams of pure cupric sulphate in distilled water, and dilute up to 500 c.c.

Second solution: Dissolve 180 grams of pure Rochelle salt and 70 grams of caustic soda in 400 c.c. of distilled water, and heat to boiling; on cooling, make up to 500 c.c. with distilled water.

To about ten minims of each solution in a test-tube add about a fluidram of distilled water, and boil for a few seconds; if the solution remains clear, add the suspected urine drop by drop, and occasionally heat the tube. If sugar is abundant, a yellowish-red deposit will be produced. If no precipitate falls, continue the addition of the urine until an equal volume has been added, and allow to cool; then if no precipitate falls, sugar is absent.

The Phenylhydrazin Test.—Put in a test-tube half filled with water phenylhydrazin (hydrochlorate) 2 grains and sodium acetate 3 grains. Dissolve by heating. Fill the tube with suspected urine, and stand in boiling water for twenty minutes. Then place in cold water. On cooling, yellow, radiating groups of needle-shaped crystals of phenylglucosazon fall, which may be detected under the microscope.

Böttger's Test.—Add to a couple of drams of suspected urine which is free from albumin an equal volume of liquor potassæ and a few grains of subnitrate of bismuth, and boil; if sugar is present, it will reduce the salt of bismuth to black metallic bismuth. Substances containing sulphur, like albumin, yield a similar black precipitate.

The Fermentation Test.—Fill a four-ounce bottle three parts full of urine, and add a fluidram of ordinary yeast or a small portion of compressed yeast; lightly cork, and subject to a temperature of 70° to 80° F. for ten or twelve hours. If sugar is present, fermentation results with the evolution of carbon dioxid, and the specific gravity of the urine falls.

Quantitative Tests.—Fermentation test: Employ two bottles of urine, and to the one add the yeast; at the end of twenty-four hours take the specific gravity of each specimen. Every degree lost in the fermented urine indicates a grain of sugar to the fluidounce.

Fehling's Test.—To 1 c.c. of Fehling's solution add 4 c.c. of distilled water, and boil; if the solution still remains clear, add $\frac{1}{10}$ c.c. of the urine from a graduated pipet, and gently heat. Continue the addition of the urine, little by little, until all blue color has disappeared. If 1 c.c. of urine has been added, it will have contained half of 1 per cent. of sugar. If 2 c.c. are used, it will have contained $\frac{1}{4}$ per cent. If but half of a cubic centimeter is used, it will have contained 1 per cent.

If the specific gravity indicates that the amount of sugar is great, dilute the urine with a definite amount of water, and estimate accordingly (Tyson).

Albuminuria.—Albumin in the urine.

Causes.—It occurs—(1) In all forms of nephritis; (2) in congestion of kidneys from diseases of the heart, lungs, and liver; (3) in conditions profoundly affecting the blood, as pernicious anemia, leukemia, purpura, and poisoning by many drugs; (4) in acute febrile diseases; (5) often in pregnancy; (6) occasionally in certain persons in health, as in young adults after exertion, or a diet rich in proteids (cyclic albuminuria), or even upon changing to the upright position (orthostatic albuminuria); (7) when the urine contains pus or blood (accidental albuminuria); (8) in many nervous diseases, as apoplexy, cerebral concussion, tetanus, epilepsy.

Tests for Albumin.—Heller's Test.—Pour a small quantity of colorless nitric acid in a test-tube, and allow an equal quantity of filtered urine to trickle from a pipet down the side of the tube and to come in contact with the acid. If albumin is present, a sharply defined white ring is formed at the line of junction.

Turpentine, copaiba, and other oleoresins eliminated in the urine yield similar rings, but the latter are redissolved on the addition of alcohol.

Uric acid produces an undefined pink ring, but it is not exactly at the line of contact, and is redissolved on the application of heat.

Johnson's Test.—Fill a six-inch test-tube two-thirds full of filtered urine, and allow a couple of drams of a clear, saturated solution of picric acid to flow down the side of the tube and to mix with the urine. Turbidity indicates the presence of albumin, and it increases on gently heating the tube near its mouth. Certain substances in the urine, like the alkaloids, produce a similar turbidity, but this disappears on the application of heat.

Roberts's Nitric Magnèsium Test.—This test is very delicate and reliable. The test-fluid is made by adding one volume of strong nitric acid to five volumes of a saturated solution of sulphate of magnesium, and is employed in the same manner as nitric acid in Heller's test.

Acetonuria.—Acetone is probably derived chiefly from the fats through the intermediary stages first of β-oxybutyric acid and then of diacetic acid. It occurs in the urine—(1) To a very slight extent in health; (2) in diabetes mellitus; (3) in starvation; (4) in chloroform narcosis; (5) in some cases of carcinoma; (6) in certain digestive disturbances.

Legal's Acetone Test.—To 4 c.c. of urine, rendered alkaline with liquor potassæ, add a few drops of a strong solution of sodium nitroprussid. If the red color produced turns purple on the addition of a few drops of concentrated acetic acid, acetone is present.

Diaceturia.—Diacetic acid is found in the urine under the same conditions as acetone. The occurrence of diaceturia in diabetes is favored by a too rigorous meat diet.

Test for Diacetic Acid.—Add a solution of ferric chlorid to urine that has not been boiled. If diacetic acid is present, a Burgundy-red color develops.

Beta-Oxybutyria.—Beta-oxybutyric acid is found often in the urine with diacetic acid. It is thought to be the cause of diabetic coma.

Hematuria.—Blood in the urine. The chief causal conditions are: (1) Traumatism; (2) acute inflammation of any part of the genito-urinary tract—kidneys, bladder, urethra; (3) calculi in the bladder or kidney; (4) congestion of the kidneys from chronic heart, lung, or liver disease; (5) conditions seriously affecting the blood, such as the specific fevers, scurvy, malaria, pernicious anemia, etc.; (6) tumors and tubercle of the kidney or bladder; (7)

varicose veins at the neck of the bladder (occasionally seen in old persons); (8) vicarious menstruation (very rare); (9) parasites in the genito-urinary tract, such as the Filaria sanguinis hominis and Distoma hæmatobium.

Diagnosis.—By the color of the urine and by microscopic and spectroscopic examination.

Heller's Test.—Boil the urine with a solution of caustic potash: phosphates are precipitated, which assume a red color from the freed hematin.

Source of Hemorrhage.—*Urethra.*—The urine first passed is bloody, and the other symptoms point to the urethra.

Bladder.—Bleeding often at the end of micturition and other symptoms point to the bladder.

Kidney.—Blood intimately mixed. There may be blood-casts or clots, and the other symptoms point to the kidneys.

Hemoglobinuria.—Blood-pigment in the urine. The chief causal conditions are—(1) Blood disintegration from acute infections (malaria, typhoid fever, yellow fever), scurvy, purpura, or poisons (potassium chlorate, carbolic acid, etc.); (2) absorption of hemorrhagic effusions and the transfusion of blood; (3) some cases of Raynaud's disease. It occasionally results from exposure or overexertion (*paroxysmal hemoglobinuria*).

Indicanuria.—Indican, or potassium indoxyl sulphate, is a product of indol derived from the bacterial decomposition of proteids in the intestine. It does not color the urine, but by oxidation it is converted into indigo-blue. It is a constituent of normal urine. It is increased (1) in all conditions which favor putrefaction in the upper bowel, as obstruction in the small intestine, acute and chronic peritonitis, typhoid fever, intestinal catarrh, and obstructive jaundice; (2) conditions associated with the decomposition of pus, as empyema, abscess, and gangrene of the lung.

Tests for Indican.—Mix equal volumes of urine and fuming hydrochloric acid, and with constant shaking, add a fresh, saturated solution of calcium hypochlorite, drop by drop, until the blue color ceases to deepen, then shake with chloroform. The latter dissolves the indigo and separates as a blue liquid, the color of which is more or less deep, according to the amount of indican.

Choluria.—The presence of bile-pigments and bile acids in the urine. It is most marked in obstructive jaundice, but it may also occur in the non-obstructive form. The urine varies from a greenish-yellow to a dark-brown color.

Tests for Bile.—Gmelin's Test.—Allow a few drops of urine and a few drops of fuming nitric acid to come together on a white plate. If bile is present, there will be an iridescent play of colors—green, blue, violet, and red—at the line of contact.

Pettenkofer's Test.—Add a few grains of cane-sugar and a drop of sulphuric acid to the suspected urine in a test-tube; heat gently, and if bile acids are present, a violet-red color is produced.

Hay's Test.—If a very small amount of flowers of sulphur be sprinkled upon the surface of the urine, it will at once begin to fall to the bottom if the slightest traces of bile are present.

Chyluria.—Chyle in the urine. The urine presents a milky appearance. The emulsion of the fat is so complete that microscopic examination rarely reveals distinct oil-globules. Ether dissolves the fat and renders the urine clear. Chylous urine is often slightly pink from the admixture of blood. The chief cause of chyluria is the obstruction of the lymphatic ducts by the Filaria sanguinis hominis.

Pyuria.—Pus in the urine. It results (1) from suppurative inflammation of any part of the genito-urinary tract, and (2) from the rupture of abscesses into the tract.

It appears as a dull, greenish-yellow precipitate that is converted into a clear gelatinous mass by the addition of liquor potassæ. It can always be detected by the microscope.

Source.—When pus is from the kidney, it is intimately mixed with the urine; the latter has an acid or neutral reaction, and the associated symptoms point to the kidneys.

When the pus is from the bladder, it is not so intimately mixed with the urine; the latter is often alkaline in reaction, and the associated symptoms point to the bladder.

Ehrlich's Diazo-reaction.—In certain diseases the urine contains aromatic bodies that produce a characteristic color with sulpho-diazobenzol.

Process.—Two solutions should be prepared and kept in separate bottles:

1.	Sulphanilic acid	5.0	2.	Sodium nitrite . . .	0.5
	Hydrochloric acid, pure	50.0		Distilled water . .	100.0
	Distilled water	1000.0			

In order to apply the test, 50 c.c. of No. 1 are added to 1 c.c. of No. 2. The mixture is added to the urine in a test-tube in the proportion of half urine and half mixture. One c.c. of ammonia-water is then added and the test-tube is violently shaken. The reaction is positive only when the resulting froth acquires a rose-red (not brown) color.

The diazo-reaction is commonly present in typhoid fever. Its value in diagnosis is lessened by its frequent occurrence in tuberculosis, measles, pneumonia, and septic diseases.

FLOATING KIDNEY.

(Movable Kidney; Nephroptosis.)

Definition.—A condition in which the kidney manifests a high degree of mobility.

Etiology.—It is much more frequent in women than in men. Tight lacing, frequent pregnancies, rapid loss of flesh, and overexertion are reputed causes. Congenital laxity of the perinephric tissues is probably the chief factor.

Symptoms.—The right kidney is the one usually affected, probably from its relation to the liver, which moves during the respiratory acts. The kidney may be found in any part of the abdomen as a movable tumor, reniform in shape, somewhat tender to the touch, and rarely imparting the pulsation of the renal artery. Not infrequently gastroptosis and enteroptosis coexist.

There may be no subjective symptoms. In many cases, however, there is a sense of discomfort in the abdomen, accompanied by digestive disturbance, hysteric manifestations, and hypochondriasis. Occasionally painful paroxysms occur simulating renal colic (Dietl's crises). These have been attributed to engorgement of the kidney from twisting of the renal vessels.

Diagnosis.—The reniform shape of the tumor, its free mobility, its stationary size, the lessened resistance on percussion over the renal region of the affected side, and the absence of cachexia will serve to diagnose a floating kidney from other abdominal tumors.

Treatment.—In many cases regulation of the diet, the avoidance of undue exertion, and the application of a broad abdominal binder will suffice. When the distressing symptoms persist and prove disabling, nephrorrhaphy (stitching the kidney to the posterior abdominal wall) should be considered.

HYPEREMIA OF THE KIDNEYS.

Varieties.—(1) Active hyperemia; (2) passive hyperemia.

ACTIVE HYPEREMIA.

Etiology.—It may be due to exposure to cold; to poisons (cantharides, turpentine, copaiba, etc.); to the acute fevers; or to pregnancy.

Pathology.—The kidneys are swollen, red, and bleed freely on section. Microscopic examination reveals engorgement of the capillaries and cloudy swelling of the epithelium.

Symptoms.—The urine is scanty and may contain a small amount of albumin and a few hyaline casts. There is no edema.

Treatment.—The patient should be kept in bed for a few days. The diet should consist of milk. Wet-cups and hot applications over the kidney region afford relief. Saline purges and vapor-baths are efficacious.

PASSIVE HYPEREMIA.

Etiology.—It is most commonly caused by chronic heart and lung diseases which impede the circulation. It may be due to pressure on the renal veins by tumors, ascitic fluid, or the pregnant uterus. It occasionally results from thrombosis of the renal veins or inferior vena cava.

Pathology.—The kidneys are swollen, firm, and of a dark-red color. The Malpighian bodies are distinctly visible. Long-standing passive congestion leads to pigmentation and to hyperplasia of connective tissue (*cyanotic induration*).

Symptoms.—The urine is scanty, dark, and of high specific gravity (1030 to 1035). It contains a small amount of albumin and often a few hyaline casts and a few red blood-cells. Symptoms of the primary disease which has caused the general venous stasis (dyspnea, cyanosis, and edema) are often superadded. Uremia does not occur.

Prognosis.—This depends upon the gravity of the primary disease.

Treatment.—The patient should be kept at rest. The diet should consist chiefly of milk and farinaceous food. Dry cupping is of service. As a diuretic, infusion of digitalis (2 to 4 drams) is distinctly useful. Hydragogue cathartics (salines and compound jalap powder) may be employed as adjuvants. In chronic heart disease such a pill as the following may be of service:

℞. Pulveris digitalis	
Pulveris scillæ	aa gr. xx
Extracti nucis vomicæ	gr. iv
Massæ ferri carbonatis	gr. xxx.—M.

Fiant in pilulæ No. xx.
SIG.—One pill four times a day.

UREMIA.

Definition.—The name applied to a group of symptoms resulting from the retention of poisons in the blood which should have been eliminated by the kidneys.

Symptoms.—It may develop slowly or abruptly in any form of nephritis, and may be manifested by any of the following phenomena: Headache, vertigo, delirium, epileptiform convulsions, coma, sudden blindness (unassociated with any retinal change), and transient paralysis from congestion or edema of the brain or spinal cord.

Pulmonary Symptoms.—Dyspnea (uremic asthma), Cheyne-Stokes breathing.

Gastro-intestinal Symptoms.—Hiccup, obstinate vomiting, and purging.

General Symptoms.—The skin is dry, the breath has a urinous odor, the pulse is slow and often of high tension, and the urine is scanty or suppressed. The temperature is usually normal or subnormal, but slight fever is not uncommon.

Diagnosis.—The urinous odor of the breath, the scanty urine, the decreased urea excretion, and the associated symptoms of nephritis will usually lead to a correct diagnosis. The differentiation of uremic coma from other forms of coma is considered on page 376.

Prognosis.—This is always grave. Recovery is possible, however, even after the most severe symptoms.

Treatment.—The chief indication is to favor elimination. Two drops of croton oil, diluted with olive oil or glycerin, or $\frac{1}{6}$ grain of elaterium, should be given at once. Sweating should be promoted by hot-air or vapor baths and the hypodermic administration of pilocarpin. If coma or convulsions appear, and the patient is not too feeble, venesection may be practised, the removal of from 15 to 20 ounces of blood sometimes exerting a very happy effect. In children a few ounces of blood may be abstracted from the loins by means of wet-cups.

After the blood has been withdrawn, normal saline solution may be injected subcutaneously. Rectal irrigation with hot saline solution is another potent measure. Convulsions may be controlled by chloroform inhalations or by an enema of chloral ($\frac{1}{2}$ to 1 dram). Morphin has been recommended, but it should be used with great caution, especially in chronic interstitial nephritis.

ACUTE NEPHRITIS.

(**Acute Bright's Disease; Acute Tubular Nephritis; Acute Desquamative Nephritis; Acute Parenchymatous Nephritis; Acute Catarrhal Nephritis.**)

Definition.—An acute inflammatory disease of the kidney, involving especially the epithelium of the tubules and glomeruli.

Etiology.—The chief causes are: (1) Infectious diseases, especially scarlet fever; (2) poisons which are eliminated through the kidneys, such as cantharides, turpentine, etc.; (3) exposure to cold and wet; (4) pregnancy. Inflammatory skin diseases and extensive burns may also cause acute nephritis.

Pathology.—The kidney is swollen and the capsule non-adherent. At first the organ is bright red in color; it soon, however, becomes pale and mottled in appearance, although the Malpighian tufts still retain their deep-red tint.

Histology.—The epithelium of the tubules and glomeruli is the seat of cloudy swelling and, later, of fatty degeneration. Desquamated epithelium, blood-corpuscles, and an albuminous exudate block the tubules. The capillaries are dilated, their walls are degenerated, and bloody extravasations are not infrequently seen. The interstitial tissue is more or less infiltrated with leukocytes.

Symptoms.—The general symptoms are moderate fever and its associated phenomena; dull lumbar pain; nausea and vomiting; dropsy, beginning in the face and becoming general; and pronounced anemia. Uremic symptoms may develop at any time.

The Urine.—The urine is scanty and at times suppressed. It is smoky in appearance, of high specific gravity, and contains a large amount of albumin, free blood, hyaline, blood, and epithelial casts, and epithelial cells. Granular casts may also be found. The daily secretion of urea is decreased.

Diagnosis.—As the general symptoms are often slight, the diagnosis must rest on the examination of the urine. The history and the absence in the urine of wide, highly fatty casts will serve to distinguish *acute nephritis* from an *acute exacerbation of chronic parenchymatous nephritis.*

Prognosis.—Guardedly favorable. It may kill by exhaustion, uremia, or edema of the lungs. It may become chronic. The average duration is from two to six weeks.

Treatment.—Absolute rest in bed for from four to six weeks is imperative. Milk largely diluted with carbonated water, Vichy, or lime-water is the best food. Beef-tea and broths should be interdicted.

In the absence of any direct remedies the indications are to divert the blood from the inflamed kidneys, to lessen their work as much as possible by increasing the action of the bowels and skin, and to meet the symptoms as they arise.

At the onset, if there be pain or suppression of urine, dry cupping, or, in severe cases, wet cupping over the region of the kidneys is of value. Following the cupping warm poultices may be applied to the loins with advantage. Cantharides, turpentine, or similar drugs are not to be used.

The bowels should be kept freely opened by means of daily purges, the best being salines in concentrated solution and compound jalap powder (20 to 30 grains).

Free sweating is very useful in promoting elimination by the skin. It may be effected by means of hot-water baths, hot packs, vapor-baths, hot-air baths, or the subcutaneous administration of pilocarpin ($\frac{1}{10}$ to $\frac{1}{6}$ grain).

Unirritating diuretics, like digitalis and potassium citrate or acetate, are useful. These drugs may be combined, as in the following formula:

℞. Potassii acetatis . . . . ʒij
Infusi digitalis . . . . f℥iij.—M.
SIG.—A tablespoonful, well diluted, thrice daily.

Excessive *dropsy* may demand puncture of the swollen parts, a free incision at the outer side of each ankle, or the insertion, beneath the skin, of delicate silver cannulæ (Southey's tubes). Uremia will call for its appropriate treatment (see p. 113). After the acute symptoms have subsided, iron may be employed in the form of Basham's mixture (2 to 4 fluidrams) to combat anemia.

CHRONIC PARENCHYMATOUS NEPHRITIS.

(Chronic Catarrhal Nephritis; Large White Kidney.)

Etiology.—It may follow acute nephritis or it may be chronic from the beginning. Habitual exposure, abuse of alcohol, chronic infections (tuberculosis, malaria), and passive congestion are predisposing factors. It occurs most often between the ages of twenty and forty.

Pathology.—In the early stages the kidney is enlarged and of a yellowish color. The capsule strips easily (*large white kidney*). Microscopically, the epithelium of the tubules and Malpighian bodies shows advanced fatty degeneration. The connective tissue is somewhat proliferated. Hemorrhagic extravasations are frequently seen. In the second stage (*fatty contracting kidney*) the organ is small and pale; its surface is uneven, and its capsule is somewhat adherent. The reduced size depends on destruction of the renal epithelium and the contraction of the overgrown connective tissue.

Symptoms.—The symptoms usually develop insidiously and consist in progressive weakness, marked anemia, dropsy (often first noted in the face on rising in the morning), digestive disturbances, and sooner or later a moderate degree of cardiac hypertrophy with high arterial tension and accentuation of the second aortic sound. Uremic symptoms may develop at any time.

The Urine.—The urine is usually diminished in quantity, is often turbid, is of rather low specific gravity, is highly albuminous, and contains wide dark granular casts, fatty casts, waxy casts, and fatty epithelial cells.

Complications.—These are numerous and often suggest the diagnosis. The most common are uremia, extensive serous effusion into the tissues or serous cavities, latent inflammation of the serous membranes, pneumonia, valvular heart disease, albuminuric retinitis, apoplexy, and acute exacerbations.

Prognosis.—Unfavorable. The duration is from a few months to several years.

Treatment.—The treatment is largely dietetic and hygienic. Residence in a dry, warm, and equable climate serves to prolong life. Rest is an essential element in the treatment. The underclothing should be woollen or silk. The diet should be non-nitrogenous, and in severe cases an absolute milk diet may be of extreme value. Warm baths with friction are useful in promoting free action of the skin, but great care must be exercised after their use to avoid chilling. The bowels should be kept active by natural mineral waters or saline laxatives. When the urine is scanty, digitalis, caffein, potassium citrate, or theobromin may prove efficacious. The following combination sometimes acts happily :

℞.	Sparteinæ sulphatis	gr. vj
	Caffeinæ citratæ	gr. xxx
	Lithii benzoatis	ʒj.—M.

Fiant chartulæ No. xij.
SIG.—One powder four times a day.

Basham's mixture is often useful as a hematinic, but only small doses should be used. Strychnin and the simple bitters are valuable adjuvants to iron in many cases. Excessive dropsy will call for hydragogue cathartics (Epsom salts, compound jalap powder, or elaterium), for diaphoretics (hot-air baths and pilocarpin), and perhaps for operative measures (puncture of the legs and scrotum, insertion of Southey's tubes, incisions near the ankles, aspiration of serous sacs). Uremia will demand special treatment (see p. 113); acute exacerbations should be treated as primary attacks of acute nephritis.

CHRONIC INTERSTITIAL NEPHRITIS.

(**Red Granular Kidney; Contracted Kidney; Gouty Kidney.**)

Definition.—A chronic inflammatory disease of the kidney characterized by a marked overgrowth of its connective-tissue elements, and almost invariably associated with general arteriosclerosis and hypertrophy of the heart.

Etiology.—It is much more common in males than in females, and is most frequently encountered between the ages of forty and sixty. (1) It is frequently a sequel of gout, chronic rheumatism, alcoholism, chronic plumbism, or syphilis; (2) it is a common accompaniment of arteriosclerosis; (3) it may follow passive congestion, as from chronic heart disease.

Pathology.—The kidney is small and red in color. The surface is granular and the capsule adherent. The organ is firm, cuts with difficulty, and on section often reveals small cysts or calcareous deposits. The cortical substance is greatly reduced in thickness. Microscopic examination shows an overgrowth of connective tissue, which in contracting has partially destroyed the glomeruli and narrowed the lumen of the tubules. The epithelium also is more or less atrophied and degenerated. The arteries throughout the body are the seat of sclerotic changes, in consequence of which hypertrophy of the heart, especially of the left ventricle, has resulted.

Symptoms.—The symptoms develop most insidiously.

There is slow loss of strength, with increasing anemia. Gastric disturbances are common. Vascular symptoms are prominent, and include thickening of the vessels, high arterial tension, accentuation of the second aortic sound, and hypertrophy of the heart. Dyspnea is present in the late stages, and may result from cardiac weakness, edema of the lungs, or uremia. Headache, vertigo, and insomnia often result from the disturbed circulation or from uremia. Dimness of vision from albuminuric retinitis is a serious symptom. Dropsy is often absent, or is slight and late in appearing. Uremia is of very frequent occurrence.

The Urine.—The urine is very copious (80 to 150 ounces), pale in color, of low specific gravity,—1005 to 1012,—and contains but a trace of albumin and few narrow hyaline or pale granular casts.

Complications.—Albuminuric retinitis, valvular heart disease, apoplexy resulting from the weakened arteries and large heart, uremia, latent inflammation of serous membranes, pneumonia, and bronchitis.

Diagnosis.—**Chronic parenchymatous nephritis** usually occurs in younger subjects and runs a shorter course. There is decided edema, and the urine is decreased in quantity and contains much albumin and wide fatty casts.

Prognosis.—The disease is incurable, but may last many years. The possibility of uremia occurring suddenly must be borne in mind.

Treatment.—The dietetic and hygienic treatment is that of chronic parenchymatous nephritis. Frequent tepid baths with friction of the skin are advantageous. The bowels should be kept regular with mild saline cathartics or alkaline mineral waters. Nitroglycerin ($\frac{1}{150}$ to $\frac{1}{50}$ grain) is often useful when arterial tension becomes excessive and causes headache, vertigo, palpitation, and dyspnea. Basham's mixture in small doses (1 fluidram) is sometimes of service when there is pronounced anemia. When there is severe insomnia, bromids, chloral, paraldehyd, and trional may be tried in the order named. Opium should be avoided. Heart-failure with low arterial tension will require the use of such stimulants as digitalis, strychnin, caffein, and alcohol.

AMYLOID DEGENERATION OF THE KIDNEY.

(Waxy Kidney; Lardaceous Kidney.)

Etiology.—It occurs in prolonged suppurative diseases, especially of bones, in tuberculosis, syphilis, and cachectic states.

Pathology.—The kidney is enlarged, firm, and pale, and on section may present a waxy, translucent appearance. The amyloid areas are colored mahogany-brown by the application of Lugol's solution to the cut surface. Other organs, especially the liver and spleen, usually share in the degenerative process.

On microscopic examination the walls of the blood-vessels, especially of those of the Malpighian bodies, are found thickened and infiltrated with a homogeneous wax-like material that turns pink when treated with gentian-violet. The epithelium is often fatty.

Symptoms.—Most patients appear badly nourished and anemic. Dropsy is present in many cases. The liver and spleen are usually enlarged from the same cause. Uremia is very rare.

The Urine.—The urine is *increased* in quantity, is rich in albumin, and is of low specific gravity. Microscopically, it contains hyaline and waxy tube-casts and degenerated epithelium.

Diagnosis.—This is based upon the history, the enlargement of the liver and spleen, and the polyuria with marked albuminuria.

Prognosis.—In the majority of cases the prognosis is very grave. In the early stages an arrest of the process is not impossible if the original disease can be cured.

Treatment.—The treatment is chiefly that of the primary disease. In other respects it must be purely hygienic, dietetic, and symptomatic.

PYELITIS.

Definition.—Inflammation of the pelvis of the kidney.

Etiology.—(1) It may result from a stone in the pelvis of the kidney (calculous pyelitis). (2) It may be secondary to urethritis and cystitis. (3) It may be tuberculous or cancerous. (4) It may be excited by irritant diuretics—cantharides, turpentine, etc. (5) It may occur in the course of specific fevers. (6) It is rarely the result of exposure to cold and wet.

Pathology.—The mucous membrane is swollen, injected, and covered with desquamated epithelium and mucus or mucopus. In severe cases the suppurative inflammation may extend to the substance of the kidney (*pyelonephritis*). In calculous and tuberculous pyelitis, especially when the ureter is obstructed, the pelvis of the kidney may become greatly distended from the accumulation of pus (*pyonephrosis*). In such cases the pus is occasionally discharged into the perinephric tissues, and ultimately even into the colon or other neighboring organs.

Symptoms.—In *simple catarrhal pyelitis* the chief symptoms are dull pain over the kidney and the passage of turbid, acid urine, containing mucus, epithelial cells, and pus-corpuscles. In *severe suppurative cases* the kidney region is often distinctly painful and tender. A tumor or swelling can sometimes be detected. Symptoms of sepsis—irregular fever, profuse sweats, chills, leukocytosis, and pallor—are frequently present. The urine is usually acid in reaction and contains more or less pus, mucus, blood, albumin, and desquamated pelvic epithelium.

Diagnosis.—In **cystitis** pain is referred to the hypogastric region, there is frequent micturition with dysuria, and the urine is more likely to be alkaline in reaction than acid.

In **perinephritic abscess** the lumbar swelling is usually more circumscribed; the superficial tissues are often edematous; and the urine is free from pus.

Calculous Pyelitis.—Sharp pain, increased by jarring movements, and reflected down the ureters, and the presence of much blood in the urine point to calculous pyelitis.

Tuberculous pyelitis may be recognized by the history, the presence of tuberculous foci in other organs, and the discovery of tubercle bacilli in the urine. The tuberculin test may also aid in the diagnosis.

Prognosis.—Mild forms resulting from exposure or the specific fevers usually recover in a few weeks. In suppurative pyelitis the prognosis is grave, although recovery may occur under operative treatment.

Treatment.—The patient should be kept in bed and placed upon a milk diet. In acute cases warm applications are useful. Alkalis and alkaline mineral waters are of service. Such a combination as the following may be prescribed:

℞.	Sodii bromidi	
	Sodii bicarbonatis	āā gr. clx
	Extracti belladonnæ	gr. iv
	Extracti buchu	ʒj
	Syrupi sarsaparillæ compositi	q. s. ad f℥iv.—M.

SIG.—A teaspoonful in water three or four times a day.

Urotropin (5 grains) or salol (3 to 5 grains) may be given for its antiseptic effect. Calculous pyelitis will require the treatment indicated for renal calculus. In suppurative cases operative interference offers the only hope of saving life.

NEPHROLITHIASIS.

(**Renal Calculus; Gravel.**)

Definition.—Renal calculi are concretions formed in the kidney by the precipitation of various solid constituents of the urine.

Etiology.—The disease is more common in males than in females. Heredity and sedentary habits are given as predisposing causes. The formation of stones is favored by the presence in the urine of any sparingly soluble substance in excess. Mucus, blood, pus, or epithelium may furnish the nucleus.

Pathology.—The size of renal concretions varies from that of coarse sand ("gravel") to that of a large bean. The most common forms are those composed of—(1) Uric acid and its compounds; (2) oxalate of lime; (3) phosphate of calcium and of ammoniomagnesium phosphate. Stones composed of xanthin and cystin are rare.

Uric acid are the most common calculi. They are usually smooth, of a reddish-brown color, and comparatively hard,

Oxalate-of-lime calculi are very hard, of a dark-brown color, and uneven (mulberry calculi). **Phosphatic calculi** are grayish-white in color, soft, and mortar-like.

Events.—(1) Small particles are frequently passed without serious disturbance. (2) Larger concretions may be extruded with intense pain (*renal colic*). (3) Calculi may remain in the pelvis and excite pyelitis or pyelonephritis. (4) They may obstruct the ureter and cause hydronephrosis or pyonephrosis.

Symptoms.—*Pain and tenderness* in the kidney region are common symptoms. The pain is aggravated by rough motion, and tends to radiate along the ureter. The urine frequently contains *blood*, *pus*, *epithelium*, and *crystals* indicating the nature of the stone.

Symptoms of Sepsis.—Irregular fever, chills, sweats, leukocytosis, and pallor mark the occurrence of suppurative pyelitis. *Colic* is excited by the entrance of the stone into the ureter.

Renal colic is characterized by intense pain radiating from the kidney downward into the groin, thigh, and testicle. The testicle is often retracted. There are often nausea, vomiting, and collapse. After such an attack the urine may contain blood or particles of stone.

Diagnosis.—In biliary colic the pain radiates to the shoulder; there is often jaundice; the gall-bladder is usually tender and enlarged; the urine is negative; a stone may be found in the stools.

Prognosis.—In uncomplicated cases the prognosis should be guardedly favorable.

Treatment.—This should be directed to the underlying diathesis. In cases of uric-acid calculi alkalis and alkaline mineral waters are useful. A quart of water containing 40 grains of potassium bicarbonate and 20 grains of lithium citrate may be taken daily. Special remedies, like piperazin, lycetol, and urosin, have been recommended as solvents, but they are of doubtful value. When phosphatic calculi are present, benzoic or boric acid (5 to 15 grains thrice daily) may be employed in a similar manner.

Operation (nephrolithotomy, nephrotomy, or nephrectomy) is urgently demanded when the attacks of renal colic occur with such frequency as to prove disabling, when there are evidences of severe pyelitis, or when there is reason to believe that the calculus has become permanently impacted in the ureter.

Renal Colic.—The indications are to relieve the pain and to relax the spasm. This is best accomplished by hypodermic injections of morphin and atropin, coupled with hot baths or local applications—hot poultices or fomentations. If the pain is extreme, it may be desirable to administer chloroform. Simple diluents should be given freely. In mild attacks full doses of phenacetin or antipyrin, with an abundant supply of hot drinks, may suffice.

HYDRONEPHROSIS.

Definition.—Dilatation of the pelvis of the kidney by retained secretion.

Etiology.—The chief causes are: (1) Congenital stricture of the ureter. (2) Impaction of a calculus in the ureter. (3) Abdominal tumors compressing the ureter. (4) Tumors growing within the urinary passages. (5) An inflammatory stricture of the ureter or urethra.

Pathology.—The pelvis reveals all grades of distention. In extreme cases it may contain several quarts of fluid, which is at first urinous, but later thin and watery. There is more or less atrophy of the renal tissue.

Symptoms.—Slight distention yields no symptoms. In other cases a tumor slowly develops in the region of the affected kidney. On palpation it is elastic, and perhaps fluctuating; on percussion, dull; and on aspiration it yields a clear fluid, which usually contains urea and uric acid.

Diagnosis.—This should be based on the history, the exclusion of other abdominal enlargements, and the chemical analysis of the fluid obtained by aspiration.

Prognosis.—When the disease is unilateral and the other kidney secretes a normal amount of urine containing a normal amount of urea, the prognosis is guardedly favorable. The disease may end fatally in consequence of rupture into the peritoneum or of secondary pyonephrosis.

Treatment.—Large accumulations will demand surgical treatment; small ones should not be disturbed.

TUBERCULOSIS OF THE KIDNEY.

Etiology.—The etiology of renal tuberculosis is that of tuberculosis in general. Males are more frequently attacked than females. The majority of cases are encountered between the ages of twenty and forty years.

Pathology.—Two forms of renal tuberculosis have been recognized—the miliary and the caseous. The former is nearly always bilateral, is an acute process, and is generally unmistakably secondary to tuberculosis elsewhere in the body. The caseous variety runs a chronic course; it usually begins as a unilateral affection, although the other organ is commonly ultimately involved, and a primary focus may or may not be apparent in some other structure.

Symptoms.—The chief symptoms are: pain in the lumbar region, usually dull, but sometimes sharp, like that of renal colic; tenderness on pressure; slight, irregular fever, and more or less cachexia. The urine is usually acid in reaction, and may contain pus, blood, albumin, tubercle bacilli, cheesy particles, and débris. Tube-casts are rarely found. In many cases enlargement of the affected organ can be detected by bimanual palpation.

Diagnosis.—**Calculous Pyelitis.**—In this condition pain is usually more severe and more apt to be affected by movement. Hematuria is more profuse, and is often excited by exertion. Cachexia is not so marked, and there are no tubercle bacilli in the urine. The tuberculin-test and the Röntgen rays may aid in the diagnosis.

Prognosis.—Always grave. Without intervention the duration is from a few months to three years.

Treatment.—When the renal disease appears to be primary and the patient's strength will permit, nephrectomy should be recommended. The mortality in operative cases has been about 28 per cent. In other cases the treatment must of necessity be palliative.

DISEASES OF THE BLOOD AND THE DUCTLESS GLANDS.

NORMAL BLOOD.

In health the blood amounts to about one-thirteenth of the body-weight. Normally there are approximately 5,000,000 red blood-corpuscles in the cubic millimeter. This number is temporarily diminished during menstruation, gestation, lactation, and fatigue, and after the ingestion of much fluid. Fasting and profuse sweating increase the number of red cells by concentrating the blood. In the first few days of life the number in a cubic millimeter may be 7,000,000 to 8,000,000. In high altitudes the number is also increased. There are from 5000 to 10,000 white cells in the cubic millimeter, the ratio of white to red cells being about 1 to 500. The number of blood-plates is from 200,000 to 300,000.

EXAMINATION OF THE BLOOD.

A clinical study of the blood has for its object the determination of the percentage of hemoglobin, the specific gravity, the alkalinity, the number, form, and relative proportion of the various corpuscles, and the detection of free pigment, bacteria, and animal parasites.

Estimation of Hemoglobin.—The percentage of hemoglobin may be determined by either Fleischl's or Gowers' apparatus, although the former is preferable.

Gowers' hemoglobinometer consists of—(1) A small sealed tube containing coloring-matter representing the color of normal blood diluted with 100 parts of water; (2) an empty tube of the same size, graduated up to 120 per cent.; (3) a small bottle with a pipet stopper, for distilled water; (4) a capillary pipet for measuring 20 cm. of blood; and (5) a small lancet. To obtain a specimen of blood the tip of the finger or the lobe of the ear, after being thoroughly cleansed, is deeply pricked with the lancet, so that the blood flows freely without squeezing; 20 cm. of blood are then drawn into the capillary pipet, and are immediately blown into the graduated tube, in which have been previously placed a few drops of distilled water to prevent coagulation. After shaking the mixture to secure diffusion of the blood, more distilled water is cautiously added, with occasional shaking, until the tint in the sealed tube is reached. The height of the column of the fluid in the graduated tube will indicate the percentage of hemoglobin.

Fleischl's instrument consists of a metal stand with a circular aperture in the center, under which is placed a reflector made of plaster-of-Paris. The aperture is fitted with a small cell having a glass bottom, and divided into two equal compartments. A graduated wedge of colored glass is employed as a standard, the 100 on the scale being intended to represent the percentage of hemoglobin in normal blood. This wedge of glass is so arranged that when moved under the stand, one compartment of the cell will receive white light from the reflector, and the other, red light from the tinted glass. A small capillary tube is held over a drop of blood until filled, and is then washed in one of the compartments of the cell, in which has been previously placed some distilled water. Both compartments are then equally filled with water, and the wedge of glass is moved by means of a thumb-screw until the tints in the two chambers are exactly the same, when the percentage of hemoglobin may be read off.

In the examination it is necessary to use artificial light. The 100 mark on the scale, which is intended to represent the percentage of hemoglobin in normal blood, is too high for the average person, 85 or 90 per cent. rarely being exceeded.

The Specific Gravity of the Blood.—The specific gravity of the blood in health varies from 1050 to 1070. In grave anemia it is often considerably diminished. Hammerschlag's method consists in expelling a drop of blood into a mixture of chloroform and benzol, one or the other of these substances being subsequently added until the drop neither rises nor falls. The specific gravity of the mixture may then be ascertained in the usual way. Lloyd Jones employs mixtures of glycerin and water of different densities, and notes the specific gravity of the mixture in which the blood-drop remains stationary.

Alkalinity of the Blood.—The alkalinity of the blood may be determined by titrating with a standard solution of acetic acid until a change of color is produced when a drop is placed on a plaster-of-Paris plate impregnated with neutral litmus.

Enumeration of Red Blood-corpuscles.—The best instrument for blood counting is the hemocytometer of Thoma-Zeiss. This consists of a glass slide in the center of which is a cell $\frac{1}{10}$ mm. in depth. The floor of the cell is divided into 400 small squares, each of which has an area of $\frac{1}{400}$ square millimeter. These small squares are grouped into sets of 16 by a series of additional vertical and horizontal lines bisecting each fifth column of squares. As the depth of the cell from the cover-glass is $\frac{1}{10}$ mm., the cubic contents of each small square is $\frac{1}{4000}$ cm.

The blood is mixed in a *mélangeur*—that is, a capillary tube one extremity of which is blown into a bulb having a capacity of 100 cm. The mélangeur is marked at 0.5, 1, and 101. A drop of blood issuing from a prick of the finger is sucked cautiously into the tube exactly to the mark 0.5. The point of the tube is quickly wiped dry and immersed in the diluting fluid (2.5 per cent. solution of potassium bichromate or Toison's fluid[1]), which is drawn up to the 101 mark. The instrument is now shaken to secure diffusion of the blood. The diluting fluid remaining in the stem of the mélangeur is now blown out, and a drop of the mixture placed upon the middle of the bottom of the divided cell. The drop in the cell should be free from bubbles, and the cover-glass so adjusted that concentric rings of color appear at the points of contact between the cover-glass and the glass plate. Before counting, a few minutes should be allowed for the corpuscles to settle to the bottom of the cell. The number of corpuscles is then counted in 400 small squares. To avoid repetition in counting, corpuscles on the upper and left boundary-lines should be counted, but those on the lower and right boundary-lines should be disregarded. The number of corpuscles in each cubic millimeter of blood is determined by multiplying the number of corpuscles counted by the degree of dilution (200) and again by the cubic contents of each square (4000), and then dividing the product by the number of squares counted (400). Thus, if 2000 corpuscles were counted in 400 squares, the number of corpuscles in each cubic millimeter would be 4,000,000 —

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[1] Methyl-violet, 5 B, 0.025 gm.; sodium chlorid, 1 gm.; pure sodium sulphate, 8 gm.; neutral glycerin, 30 c.c.; distilled water, 160 c.c.

$$\frac{2000 \times 200 \times 4000}{400} = 4{,}000{,}000.$$

After using, the mélangeur should be carefully washed in water, alcohol, and ether.

Enumeration of White Blood-corpuscles.—For counting the white blood-cells a mélangeur should be used which allows a dilution in the proportion of 1 : 10 and an aqueous 0.5 per cent. solution of acetic acid, to which may be added a little methyl-violet, should be selected as a diluting fluid. The red cells disappear in this solution, and the white cells remain and are readily counted. The latter should be counted in 800 small squares. The number of leukocytes in each cubic millimeter is then determined by multiplying the whole number counted by 4000, and again by 10, and dividing by 800.

The Study of the White Blood-corpuscles.—In normal blood the following forms of leukocytes may be recognized.

1. **Small Lymphocytes.**—These are small cells about the size of the red blood-corpuscles. The nucleus is very large and spheric, and stains intensely with basic stains (methylene-

blue). With Ehrlich's triacid mixture the nucleus is pale. The narrow rim of protoplasm surrounding the nucleus is non-granular (hyaline). Small lymphocytes constitute from 25 to 35 per cent. of all leukocytes.

2. **Large Lymphocytes.**—These cells resemble those just described, but they are considerably larger. The nucleus is relatively not so large, and stains less deeply. In some form the nucleus is more or less bent or indented (transitional leukocytes). Normally, large lymphocytes make up from 5 to 10 per cent. of the blood-corpuscles.

Polymorphonuclear Neutrophiles.—These cells are somewhat smaller than large lymphocytes, and are actively ameboid. The nucleus appears to be divided into two or more segments and stains deeply. The protoplasm is studded with fine granules, which do not stain well with either simple basic stains (methylene-blue) or simple acid stains (eosin). With Ehrlich's triacid mixture the granules are colored violet and the protoplasm pale pink. Neutrophiles make up from 60 to 70 per cent. of the white cells of normal blood.

Eosinophiles.—These resemble the polymorphonuclear neutrophiles, but are more irregular in outline, and the granules are larger, more highly refractive, more loosely attached, and have a special affinity for acid stains (eosin). Eosinophiles make up from 1 to 4 per cent. of the leukocytes.

Mast-cells (Basophiles).—These cells have a lobulated nucleus. The protoplasm is studded with granules having an intensely basic reaction. These granules remain unstained with Ehrlich's triacid mixture, but with methylene-blue they stain deep blue. Mast-cells are only occasionally encountered in normal blood.

In disease, additional forms are sometimes found. Thus in leukemia large cells are found which are non-ameboid and which have a single round or oval nucleus imbedded in protoplasm containing neutrophilic granules. These have been termed **myelocytes.**

With the aid of a one-twelfth inch oil-immersion lens large and small leukocytes can readily be distinguished in preparations of fresh blood, but to study satisfactorily the various forms it is necessary to dry and then stain the specimen.

The Drying and Staining of Blood.—A small drop of blood, secured by pricking the finger, is spread into a film by being pressed between two perfectly clean cover-glasses, which are then drawn apart and exposed to the air until dry. The cover-glasses should be handled with forceps, since the moisture of the fingers distorts the corpuscles. The preparation is first "fixed" by heating on a copper bar for from one-half to one hour at a temperature of from 100° to 110° C., or by immersing for from five to fifteen minutes in a mixture of equal parts of absolute alcohol and ether.

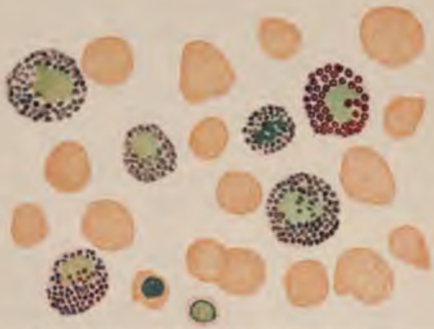

FIG. 7.—Blood in lienomedullary leukemia, showing several mononuclear neutrophiles (myelocytes), one polymorphonuclear neutrophile, and an eosinophile; a nucleated red corpuscle and a lymphocyte are seen in the lower part of the illustration. Stained with Ehrlich's triple mixture (from Stengel's *Text-Book of Pathology*).

A convenient method of staining is the one suggested by Stengel. The fixed preparation is immersed for a few minutes in a 1 per cent. solution of eosin in 60 per cent. alcohol, to which has been added an equal quantity of water at the time of staining. The cover-glass is then washed in water and counterstained in Delafield's hematoxylin for a minute, and finally washed, dried, and mounted. The eosinophile granules are dark red, the red corpuscles lighter red, and the nuclei of the leukocytes almost black. Ehrlich's triacid mixture (methyl-green, Orange G, and acid-fuchsin) makes an excellent stain. The film should be fixed by heat and flooded with the stain. After the lapse of from five to eight minutes the stain should be washed off in running water and the film dried by gentle heat and mounted in xylol balsam. With this stain the red cells are tinged orange, the nuclei of the leukocytes greenish-blue, the neutrophile granules violet, the eosinophile granules red, the nuclei of normoblasts purple, and the nuclei of macroblasts greenish-blue.

PLETHORA.

An increase in the whole quantity of blood. It is very doubtful whether such a condition can be more than transitory.

HYDREMIA.

An excess of water in the blood. As a loss of corpuscular elements is generally replaced by the addition of water extracted from the tissues, most anemias are associated with hydremia. The condition is more marked in general dropsy. Temporary hydremia is produced by the excessive ingestion of fluids.

ANHYDREMIA.

A deficiency of fluid in the blood. It is observed in starvation, immediately after hemorrhage, and after copious discharges, as in cholera.

MELANEMIA.

A condition in which free pigment granules occur in the blood. It is met with in malaria and certain other fevers, and occasionally in melanosarcoma and in Addison's disease. The pigment may be found in the plasma or in the leukocytes.

POLYCYTHEMIA.

Polycythemia, or an increase in the number of red cells, is an apparent condition in blood taken from cyanosed parts. It is observed temporarily in the new-born, in recovery from certain anemias, after transfusion of blood, and in blood concentrated by excessive discharges. Marked polycythemia is sometimes produced by residence in high altitudes and by certain poisons, such as phosphorus and carbon monoxid. It also occurs in the condition known as chronic splenomegalic polycythemia.

MICROCYTOSIS AND MACROCYTOSIS.

Microcytosis and macrocytosis are conditions in which the red cells are respectively diminished and increased in size. They may occur in any form of severe anemia, but they are especially marked in pernicious anemia.

POIKILOCYTOSIS.

Poikilocytosis, a condition in which the red cells are irregular in shape, is common in grave anemias, especially pernicious anemia.

NUCLEATED RED CELLS.

Nucleated red cells (erythroblasts) are divided into three forms—normoblasts, macroblasts, and microblasts. The first resemble in size and color a normal red cell, the second are larger, and the third smaller. Nucleated red cells are not found normally in the circulating blood; they are present, however, in grave forms of anemia.

LEUKOCYTOSIS.

Leukocytosis, or hyperleukocytosis, is an increase in the number of white cells, especially of the polymorphonuclear forms, in the peripheral blood. It occurs *physiologically* in the new-born, during digestion, in pregnancy, parturition, and after heavy exertion, cold bathing, and massage.

Pathologic leukocytosis is observed in the following conditions: (1) Inflammation. There is an absolute increase in the polymorphonuclear neutrophiles. (2) Infectious diseases. Most infections excite leukocytosis, but the condition is usually wanting in typhoid fever, malaria, measles, influenza, and miliary tuberculosis. In any infection in which the toxemia is intense or the resistance of the individual is slight, leukocytosis may be wanting. (3) Malignant disease, when sufficiently extensive. (4) After hemorrhage. (5) After the administration of certain drugs, such as pilocarpin, antipyrin, salicylates, ergotin, and tuberculin. (6) In certain autointoxications, such as gout and uremia.

EOSINOPHILIA.

A relative or absolute increase of the eosinophiles occurs in certain diseases caused by animal parasites, such as trichiniasis, filariasis, and ankylostomiasis; in bronchial asthma, in osteomalacia, and in certain skin diseases, notably in pemphigus, eczema, psoriasis, and dermatitis herpetiformis.

LEUKOPENIA, OR HYPOLEUKOCYTOSIS.

Leukopenia, or hypoleukocytosis, is the name applied to a deficiency in the number of leukocytes. It occurs in certain infections, particularly in those that do not produce leukocytosis, such as typhoid fever, malaria, and miliary tuberculosis; also in pernicious anemia and inanition.

LIPEMIA.

Lipemia, the presence in the blood of minute fat-globules, may be noted in health. Abnormal quantities of fat may be found in the blood in diabetes, chronic nephritis, alcoholism, and pulmonary tuberculosis.

PARASITES IN THE BLOOD.

The following parasites have been detected in the blood: Filaria sanguinis hominis, hematozoan of malaria, spirillum of relapsing fever, pneumococcus, bacillus of anthrax, typhoid fever, tetanus, tuberculosis, influenza, leprosy, glanders, bubonic plague, malignant edema, and diphtheria; the streptococcus, staphylococcus, meningococcus, gonococcus, trypanosoma, and colon bacillus.

OLIGOCHROMEMIA.

Oligochromemia, or deficiency of hemoglobin, is usually proportionate to the reduction in the number of red cells, but there are two exceptions, namely, in chlorosis, in which disease the red cells may be reduced only 20 or 30 per cent., while the hemoglobin may be reduced 50 or 60 per cent., and in pernicious anemia, in which disease the blood-count is very low, while the corpuscles are relatively rich in hemoglobin.

The *color-index* represents the relation between the number of cells and the quantity of hemoglobin. In a patient having 2,500,000 red cells per cubic millimeter (50 per cent.) and 40 per cent. of hemoglobin, the color-index would be $\frac{40}{50} = 0.8$.

OLIGOCYTHEMIA.

Oligocythemia, a diminution in the number of red cells, occurs in all forms of anemia, but it is especially marked in pernicious anemia and in advanced malignant disease, where the number may fall below 1,000,000 in a cubic millimeter.

ANEMIA.

Definition.—A deterioration of the blood, with altered relations of the fluid and solid parts (Stengel).

Varieties.—(1) Secondary anemia; (2) primary anemia.

Symptoms.—Any forms of anemia may present the following symptoms: Pallor of the skin and mucous membranes, loss of strength, and, in severe cases, febrile paroxysms.

Circulation.—A full, rapid pulse, unnatural pulsation of the cervical vessels, palpitation of the heart, a hemic murmur, a hum over the jugular vein, and slight dropsy, beginning in the feet. In severe forms there may be ecchymoses and bleeding from mucous membranes.

Respiration.—Hurried breathing.

Digestion.—Dyspepsia.

Nervous System.—Headache, vertigo, disturbed sleep, neuralgic pains, and tendency to syncope.

SECONDARY ANEMIA.

Definition.—A secondary anemia is one that is symptomatic of some conspicuous underlying condition.

Etiology.—Secondary anemia usually results from one of three causes: (1) Insufficient nutriment entering the circulation (inadequate food, chronic gastritis, cancer of the pylorus, etc.). (2) Excessive demands upon the blood-making organs (overwork, hemorrhage, chronic diarrhea, etc.). (3) Action of parasites or toxic agents (malaria, lead, syphilis, uremia, etc.).

The anemia produced by the presence in the bowel of the Ankylostomum duodenale and Bothriocephalus latus may be due to poisons generated by these parasites.

Symptoms.—In addition to the ordinary phenomena of anemia the blood-count reveals a decrease in the number of red cells and a proportionate deficiency in the percentage of hemoglobin. The number of polymorphonuclear leukocytes is often increased. In severe form, microcytes, macrocytes, and poikilocytes are present, and occasionally nucleated red cells.

Prognosis.—This depends on the cause.

Treatment.—This includes the removal of the cause, when possible; the adoption of hygienic measures; and the use of iron, arsenic, and general tonics.

PRIMARY ANEMIA.

Definition.—A primary anemia is one that, in the present state of our knowledge, cannot be associated with any conspicuous underlying cause.

Varieties.—Pernicious anemia, chlorosis, leukemia, Hodgkin's disease, and splenic anemia.

PERNICIOUS ANEMIA.

(Progressive Pernicious Anemia.)

Definition.—A grave form of anemia characterized by extreme oligocythemia, marked changes in the red blood-corpuscles, and a decrease in the number of the polymorphonuclear neutrophiles.

Etiology.—In many cases no adequate cause is apparent. The disease usually appears about middle life, and is somewhat more frequent in males than in females. Forms of anemia closely resembling pernicious anemia may result from the action of intestinal parasites, especially the Bothriocephalus latus and the Ankylostoma duodenale. The most plausible theory is that the disease is due to the hemolytic action of some poison absorbed from the gastrointestinal tract.

Pathology.—The skin has a lemon-yellow hue, the subcutaneous fat is often well preserved, and the muscles are unusually red. The organs are pigmented and fatty. Iron pigment is especially abundant in the outer zones of the hepatic lobules. Marked atrophy of the gastric mucosa is sometimes observed. The bone-marrow is dark red, soft, and contains large numbers of nucleated red cells, especially macroblasts. The hemolymph glands are frequently enlarged, congested, and pigmented. The spleen is sometimes enlarged. In many cases there is found advanced sclerosis of the posterior and lateral columns of the spinal cord.

Symptoms.—The general symptoms are intense anemia, with its usual manifestations; a lemon-yellow tint to the skin; progressive weakness, without marked emaciation; moderate, irregular fever; severe gastric irritability; and sometimes dark-colored urine from the presence of urobilin.

The Blood.—The drop is pale and watery. Coagulation is slow. There is a great reduction in the number of red cells, often to 1,000,000 or less; the hemoglobin is also reduced, but not proportionately. The red cells usually show decided changes both in size and in shape. Nucleated red cells are more or less abundant. As a rule, the large forms (megaloblasts) predominate. The leukocytes are usually decreased, though the lymphocytes are relatively increased.

Diagnosis.—The **parasitic form** may be recognized by the occurrence of eosinophilia and the discovery of the parasites or their ova in the stools.

Cancer rarely produces such extreme oligocythemia, the color-index is not high, macroblasts are rarely present, and there is often leukocytosis.

Prognosis.—Pernicious anemia usually ends fatally within one or two years. It is doubtful whether recovery ever occurs except in the parasitic forms. Periods of marked improvement are not uncommon.

Treatment.—Fresh air, rest, and a diet as liberal as the digestive power of the patient will permit are requisite. Warm salt baths and massage are valuable adjuvants to internal treatment. The teeth should receive careful attention. If there be gingivitis or pyorrhœa alveolaris, antiseptic mouth-washes should be used at frequent intervals.

Arsenic is the most valuable drug. It may be given in the form of Fowler's solution, the dose being gradually increased from 2 or 3 to 15 or 20 minims three times a day. Iron is rarely of service. Bone-marrow is sometimes efficacious. Inhalations of oxygen have been recommended (Shattuck). Appropriate anthelmintic remedies should be given, of course, in the cases in which intestinal parasites are present. Digestive disturbances are often benefited by the administration of diluted hydrochloric acid and a bitter.

CHLOROSIS.

(**Green Sickness; Primary Anemia.**)

Definition.—A form of anemia occurring exclusively in young women and characterized by marked oligochromemia.

Etiology.—The disease usually occurs between the fifteenth and twenty-fifth years. Heredity, bad hygienic surroundings, and overwork are predisposing factors. The real cause of the disease has not been determined.

Pathology.—In some fatal cases imperfect development of the vascular and generative systems has been observed.

Symptoms.—In addition to the general symptoms of anemia the conspicuous features are a greenish hue of the skin; pallor and weakness without marked loss of flesh; perversions of appetite (pica); menstrual disorders; and a tendency to hysteric outbreaks. The *blood changes* are characteristic. The number of red cells is moderately reduced (not often below 3,500,000); the hemoglobin, on the other hand, is greatly reduced—usually to below 50 per cent. There is no leukocytosis.

Complications.—Gastroptosis, peptic ulcer, gastralgia, amenorrhea, and, occasionally, thrombosis of the cerebral sinuses or veins of the extremities.

Prognosis.—The prognosis is good, but relapses are common.

Treatment.—Fresh air, sunlight, open-air exercise, and nourishing food are valuable aids in treatment. Very severe cases require complete rest in bed. If there be a good reaction, warm baths, followed by short cold douches, are efficacious. Iron is almost a specific. It is most frequently prescribed in the form of Blaud's pills, of which the dose is three pills, gradually increased to nine, a day.

Laxatives, preferably mild salines, rank next in importance to iron. Arsenic is distinctly less valuable than iron. Superacidity of the gastric juice is best treated by alkalis.

LEUKEMIA.

(Leukocythemia.)

Definition.—A disease characterized by a persistent increase in the number of white blood-corpuscles and by pathologic changes in the bone-marrow, spleen, and lymphatic glands.

Etiology.—The causes are obscure. More males are affected than females. The disease occurs most frequently in middle life. Heredity, malaria, syphilis, pregnancy, and traumatism are given as predisposing factors. An infectious origin has been suggested.

Varieties.—(1) Myelogenous leukemia and (2) lymphatic leukemia. The latter may be acute or chronic.

Pathology.—There is extreme emaciation. In the *myelogenous form* the fat of the bone-marrow is largely replaced by marrow-cells, both red and white, and, according as the one or the other predominates, the marrow presents a currant-jelly or pyoid appearance. All the cells of the marrow are increased, but the myelocytes are in excess. The spleen and liver are much enlarged: the former from a hyperplasia of the lymphoid structures, the latter from infiltration of leukocytes. In *lymphatic leukemia* the characteristic feature is enlargement of the visible lymph-glands. Microscopically, the glands show a great increase of lymphocytes. Numerous metastatic lymphomas are found in the various organs. The bone-marrow, spleen, and liver are also more or less involved.

Symptoms.—*Myelogenous leukemia* presents the general symptoms of anemia. The liver and spleen are considerably enlarged. There may be moderate fever of an irregular type. Hemorrhages from mucous membranes and into the tissues are common. Impairment of vision may result from retinal hemorrhage or leukemic infiltration. Persistent priapism is occasionally observed.

The blood changes are characteristic. There is a gradual reduction in the number of red cells and of hemoglobin. The number of white cells is enormously increased, the count often reaching from 300,000 to 500,000. From 20 to 60 per cent. of the leukocytes are myelocytes. The number of eosinophiles and mast-cells is somewhat increased.

Lymphatic leukemia is rare. The chronic form presents the general symptoms of anemia, with enlargement of the visible lymph-glands and a considerable increase in the number of leukocytes (50,000 to 100,000), more than 90 per cent. of which are small lymphocytes.

Acute lymphatic leukemia has been most frequently seen in children. The visible lymph-glands are enlarged, there is marked tendency to hemorrhages, and the blood contains great numbers of large pale lymphocytes.

Diagnosis.—**Leukocytosis.**—In this condition the white cells are not so enormously increased and are chiefly polymorphonuclear neutrophiles.

Malarial Cachexia.—This may be recognized by the discovery of the parasites and by the absence of leukocytosis.

Prognosis.—Absolutely unfavorable. The average duration of the chronic form is from two to three years. Acute leukemia may prove fatal within a few weeks.

Treatment.—An effort should be made to maintain the general nutrition by regulating the diet and attending to hygienic measures. Rest is often advisable. Among drugs, arsenic appears to be of some service. The use of the *x*-rays is often followed by marked, though temporary, improvement. Operative treatment is of no avail.

HODGKIN'S DISEASE.

(**Pseudoleukemia; General Lymphadenoma; Adenia; Lymphatic Anemia.**)

Definition.—A rare disease characterized by hyperplasia of the lymphatic glands and progressive anemia, without a marked excess of white corpuscles.

Etiology.—The causes are unknown. It is most commonly seen in young adults of the male sex. In some instances it has apparently followed an ordinary adenitis. An infectious origin has been suggested.

Pathology.—There is a marked hyperplasia of the lymphatic glands,—cervical, axillary, mediastinal, inguinal, and retroperitoneal,—the spleen and bone-marrow often sharing in the process.

Symptoms.—The disease resembles lymphatic leukemia, but there is an absence of leukocytosis. The glands of the neck are usually first affected; the swellings are painless, freely movable, and only very rarely suppurate.

Diagnosis.—Tuberculous adenitis is more apt to affect the submaxillary glands, and is often unilateral. Fusion of the glands and suppuration are common. The tuberculin reaction may be obtained, and section of an excised gland shows tuberculous elements.

Prognosis.—Unfavorable. The average duration is from two to three years. Treatment is that of leukemia.

SPLENIC ANEMIA.

(**Splenomegaly.**)

Splenic anemia is a chronic affection characterized by an enormous enlargement of the spleen, moderate oligocythemia (average count 3,400,000), decided oligochromemia (average 45 to 50 per cent.), leukopenia, and a marked tendency to hemorrhage, especially from the stomach. In the terminal stage there may be, in addition, ascites, jaundice, and cirrhosis of the liver (*Banti's disease*). Splenic anemia usually lasts for many years. In 7 of 18 cases studied by Osler the duration of the disease was more than ten years, and in 11 more than four years. Of 32 cases of splenic anemia treated by splenectomy, in 23 recovery followed (Armstrong).

CHRONIC SPLENOMEGALIC POLYCYTHEMIA.

Chronic splenomegalic polycythemia is a rare affection having general redness or cyanosis of the skin, polycythemia (8–13 millions per cmm.), high blood-pressure, and enlargement of the spleen for its chief symptoms. Later, asthenia, dyspnea, edema, dilatation of the heart, and albuminuria occur and may be associated with drowsiness and mental depression. The course extends over many years, but the tendency is to terminate ultimately by heart failure or apoplexy.

According to one theory, the disease depends upon an overproduction of the red cells in the bone marrow, which sometimes shows evidence of proliferation; according to another theory, it is the result of vasoconstriction of nervous origin.

ADDISON'S DISEASE.

Definition.—A rare disease characterized anatomically by lesions of the suprarenal glands or of the abdominal sympathetic ganglia, and clinically by bronzing of the skin and profound asthenia.

Etiology.—It occurs most frequently in middle life, and is more common in men than in women. The development of Addison's disease is favored by the predisposing causes of tuberculosis.

Pathology.—In most instances tuberculosis of the suprarenal bodies is discovered. Rarely other lesions of the suprarenal bodies, such as carcinoma, have been found. In a few cases there have been found degenerative changes in the abdominal sympathetic ganglia, either in connection with, or in the absence of, disease of the suprarenal bodies.

Symptoms.—The chief features are moderate anemia, bronzing of the skin, pigmentation of the mucous membranes, especially of the mouth, extreme weakness, and marked gastric irritability.

Prognosis.—The disease is fatal. The average duration is from one to three years.

Treatment.—The general treatment includes rest, a nutritious but easily assimilable diet, and the administration of tonics. Temporary good effects have followed the use of the extract of suprarenal gland in doses of from 3 to 5 grains thrice daily.

EXOPHTHALMIC GOITER.

(Graves's Disease; Parry's Disease; Basedow's Disease.)

Definition.—A disease characterized by enlargement of the thyroid gland, marked prominence of the eyeballs, tachycardia, and muscular tremors.

Ætiology.—The disease most frequently develops in the third decade. Women are much more often affected than men. Heredity, emotional excitement, and mental strain are predisposing factors.

Pathology.—The pathogenesis of Graves's disease is still undetermined. The disease is probably due to excessive functional activity of the thyroid gland (hyperthyroidization). Greenfield found the tubular spaces of the gland proliferated and the colloid matrix replaced by a mucoid material.

Symptoms.—**Cardiac Phenomena.**—Acceleration of the pulse (100 to 150) and palpitation are constant features. Both are intensified by excitement. Hypertrophy of the heart may ultimately ensue from overaction. A soft systolic murmur is frequently heard at the apex.

Ocular Phenomena.—These consist in bilateral protrusion of the eyeballs, a failure of the upper eyelid to follow the eyeball when the latter is directed downward (Graefe's sign), widening of the palpebral angle (Stellwag's sign), and inability of the eyes to converge upon a near object (Möbius' sign). Vision is not disturbed.

Thyroid Phenomena.—Enlargement of the thyroid may be the last symptom to appear. One or both lobes of the gland may be affected. Inspection detects enlargement, with pulsation; palpation, a purring thrill; and auscultation, a bruit.

Nervous Phenomena.—A fine muscular tremor is an early symptom. Nervous irritability and asthenia are often marked. Occasionally mania or hypochondriasis is observed.

General Symptoms.—As the disease progresses weakness and anemia become pronounced. Sweating is common. Moderate fever is an occasional symptom. There may be glycosuria and albuminuria.

Diagnosis.—It should be borne in mind that any one of the important symptoms may be absent throughout the disease. In some cases palpitation and throbbing of the cervical vessels may be the only phenomena.

Goiter may be distinguished from exophthalmic goiter by the absence of cardiac, ocular, and nervous symptoms.

Prognosis.—The disease generally runs a protracted course. Some cases recover entirely; many improve and subsequently relapse; a few die, after a short illness, from heart failure or acute mania.

Treatment.—The general nutrition should be improved by rest, a generous, readily digestible diet, healthy hygienic surroundings, and hydrotherapy. In severe cases absolute rest in bed is an essential point in the treatment. Applications of cold, by means of Leiter's tubes or ice-bags, to the precordium lessen the palpitation.

Belladonna is undoubtedly of value in many cases. It should be given in ascending doses until some dryness of the throat is produced. When the circulation is feeble, digitalis may be found of service; on the other hand, when the heart is strong, better results may be obtained with aconite or veratrum viride. When anemia exists, iron is useful. Bromids are sometimes of service in controlling nervous symptoms. Starr has observed marked improvement from the use of sodium glycerophosphate in doses of 20 grains three or four times a day. The consensus of opinion is decidedly adverse to the use of thyroid extract.

Galvanism sometimes proves more effective in controlling the symptoms than any other remedy. As a last resort, operative interference should be considered.

MYXEDEMA.

Definition.—A disease characterized by atrophy of the thyroid gland, mucoid degeneration of the subcutaneous tissues, subnormal temperature, and mental sluggishness.

Etiology.—The disease is much more frequent in women than in men. It is occasionally hereditary. It usually develops in middle life. The immediate cause is the loss of function of the thyroid gland.

A congenital form of myxedema is observed in *cretinism*, and an analogous condition (*operative myxedema* or *cachexia strumipriva*) frequently follows total extirpation of the thyroid gland.

Symptoms.—It is manifested by a gradual swelling of the subcutaneous tissues, particularly of the face, supraclavicular regions, and hands. Unlike edema, the parts do not pit on pressure, but are firm and elastic. The skin is dry and harsh. The hair is dry and brittle. The thyroid gland is atrophied. A peculiar slowness in thought, speech, and movements is a characteristic symptom. The temperature of the body is subnormal. There is impairment of the special senses. Sensory phenomena are common, such as coldness, numbness, and tingling. The urine is often increased in quantity, and occasionally contains albumin, sugar, and tube-casts.

Sequels.—Tuberculosis, dementia, and, occasionally, exophthalmic goiter.

Diagnosis.—The mental dulness, the extreme dryness of the skin, and the absence of pitting on pressure will serve to distinguish myxedema from Bright's disease with edema.

Prognosis.—The disease was formerly considered incurable, but it is now known that marked amelioration or even a cure can be effected by appropriate treatment.

Treatment.—As patients with myxedema are extremely susceptible to low temperatures, they should be warmly clad and protected from exposure to cold. Residence during the winter in a warm, sunny climate is desirable. Warm baths are often beneficial. Modern treatment consists in the administration of extract of sheep's thyroid (3 to 5 grains thrice daily). By continuing this remedy throughout life it is possible in many cases to hold the symptoms in complete abeyance.

DISEASES OF THE CIRCULATORY SYSTEM.

INSPECTION.

INSPECTION determines the position, force, and extent of the apex-beat; any abnormal centers of pulsation; and any unnatural prominence over the precordial region.

The Apex-beat.—The normal position of the apex-beat is in the fifth intercostal space, about an inch within the mid-clavicular line. The beat can usually be detected by inspection or palpation, but when these methods fail, it may be localized by auscultation, the point in the region of the apex where the first sound is heard with maximum intensity corresponding to the beat.

The Effect of Respiration and Position on the Apex-beat.—The location and force of the apex-beat are modified by the posture of the patient and the stage of the respiratory act. In the recumbent position the apex-beat may be elevated half an inch or more, and, when the body is inclined to the left, the heart being a more or less movable organ, the beat may be detected in the mammary line, or even some distance to its outer side.

During forced inspiration the beat may become imperceptible, or, if such is not the case, it may be found some distance below its usual place, on account of the upward movement of the ribs in the inspiratory act. During forced expiration the air, being driven from the lung-tissue in front of the heart, the beat becomes more forcible, and its position elevated on account of the descent of the ribs which occurs in expiration.

In view of the influence exerted by respiration and position on the apex-beat the patient, as a rule, should be examined in the erect or sitting posture, while breathing quietly.

Displacement of the Apex-beat.—*Displacement to the left* may result from:

1. Hypertrophy or dilatation of the heart (down and to the left).
2. Pericardial effusion (up and to the left).
3. Chronic diseases of the left lung and pleura, associated with retraction—as fibroid phthisis and pleural adhesions.
4. Abdominal tumors and effusions (up and to the left).
5. The pressure of a pleural effusion on the right side (up and to the left).

Displacement to the right may be caused by:

1. Chronic disease of the right lung or pleura associated with retraction.
2. Pressure of a pleural effusion on the left side.
3. Transposition of the viscera.

Displacement downward may result from:

1. Hypertrophy or dilatation of the heart, chiefly of the left ventricle.
2. Pressure of solid growths in the upper mediastinum.
3. Aneurysm of the aortic arch.

Deformity of the chest from spinal curvature may also cause considerable displacement of the heart.

Changes in Force and Extent of the Apex-beat.—*The force and extent of the pulsation may be increased* by:

1. Hypertrophy of the heart.
2. Forcible action of the heart caused by emotional or physical excitement, reflex irritation, drugs, Graves's disease, etc.
3. Thinning of the chest-walls and shrinking of the lungs, as in phthisis.

A weak apex-beat may be noted:

1. In health.
2. Degeneration or dilatation of the heart.
3. Pericardial effusion.
4. Emphysema.
5. Collapse or shock.

Abnormal Centers of Pulsation.—*Epigastric pulsation* may result from:

1. Excited action of the heart from any cause.

2. Enlargement of the right ventricle.

3. A pulsating aorta, noted in certain nervous and anemic patients.

4. Aortic aneurysm.

5. Tumors of the left lobe of the liver resting on the aorta.

Pulsation at the base of the heart may result from:

1. Aneurysm of the aortic arch.
2. Cardiac hypertrophy.
3. Shrinking of the lungs, as in phthisis.

Pulsation in the left axillary region may result from:

1. Enlargement of the heart.
2. A tense, purulent effusion in the left pleural sac (pulsating empyema).
3. Aneurysm.
4. Chronic diseases of the left lung and pleura, associated with retraction.

Unnatural pulsation in the carotids may result from:

1. Excitement of the heart from any cause.
2. Exophthalmic goiter.
3. Anemia.
4. Valvular disease, especially aortic regurgitation.
5. Aneurysm or dilatation of the vessels.
6. Unnatural elasticity of the vessels, noted in certain nervous and anemic patients.

Jugular Pulsation.—The jugular vein often becomes distended in forced expiration and coughing. Distention of the jugular vein is sometimes noted in adherent pericardium.

A true, rhythmic venous pulsation usually results from tricuspid regurgitation.

A pulsation may be transmitted to the jugular vein from the underlying carotid, but this false pulsation will still continue when light pressure is made on the vein at the root of the neck, while the true venous pulse will cease.

Precordial Prominence.—*Unnatural prominence of the precordium* may result from:

1. Hypertrophy of the heart.
2. Dilatation of the heart.
3. Pericardial effusion.

PALPATION.

This not only determines the position, force, extent, and rhythm of the apex-beat, but also detects the existence of any fremitus or thrill.

A *thrill* is a vibratory sensation likened to that received when the hand is placed on the back of a purring cat. Thrills at the base of the heart may result from aortic stenosis, atheroma of the aorta, aneurysm, and from roughened pericardial surfaces, as in pericarditis.

A presystolic thrill at the apex is almost pathognomonic of mitral stenosis.

PERCUSSION.

This determines the shape and extent of the cardiac dulness.

The normal area of superficial or absolute percussion-dulness (the part uncovered by lung) is detected by light percussion, and extends from the fourth left costosternal junction to the apex-beat; from the apex-beat to the junction of the xiphoid cartilage with the sternum, and thence up the left border of the sternum.

The normal area of deep percussion-dulness (the heart projected on the chest-wall) is detected by firm percussion, and extends from the third left costosternal articulation to the apex-beat; from the apex-beat to the junction of the xiphoid cartilage with the sternum, and thence up the right border of the sternum to the third rib. The lower level of the cardiac dulness fuses with the liver dulness, and can rarely be determined by percussion.

The area of cardiac dulness is increased in: (1) Hypertrophy and dilatation of the heart. (2) Pericardial effusion. It is apparently increased in shrinking of the lungs, as in phthisis.

The area of cardiac dulness is diminished in: (1) Emphysema. (2) Pneumothorax. (3) Pneumopericardium (rare). (4) Gaseous distention of the stomach.

AUSCULTATION.

This determines the quality, intensity, and rhythm of the heart-sounds, and detects the presence of any adventitious sounds, as murmurs. The two sounds heard over the heart have been represented by the syllables, "lubb, tup." The first sound (*systolic*) results from the muscular contraction of the heart and the closure of the auriculoventricular valves, and is synchronous with the apex-beat and carotid pulse. This sound is prolonged and dull. After the first sound there is a short pause, and then follows the second sound (*diastolic*), which results from the closure of the aortic and pulmonary valves. This sound is short and high-pitched. After the second sound a longer pause follows before the first is again heard.

The Intensity of the Heart-sounds.—*Both sounds are accentuated* in: (1) Excitement of the heart from any cause. (2) Anemia. (3) Cardiac hypertrophy. (4) Subjects with thin chest-walls. (5) Consolidation of the lung, as in phthisis and pneumonia.

Accentuation of the aortic second sound results from: (1) Hypertrophy of the left ventricle. (2) High arterial tension, as in chronic interstitial nephritis with arteriosclerosis. (3) Aortic aneurysm.

Weakening of the aortic second sound indicates weakness of the left ventricle.

Accentuation of the pulmonary second sound results from: (1) Pulmonary obstruction, as in emphysema, pneumonia, and the congestion of the lungs following mitral disease. (2) Hypertrophy of the right ventricle.

Weakness of the pulmonary sound indicates failure of the right ventricle, and, occurring in diseases in which it should be accentuated, is of grave omen.

Weakness of the mitral sound is noted in: (1) General obesity. (2) General exhaustion. (3) Degeneration or dilatation of the heart. (4) Pericardial or pleural effusion. (5) Emphysema.

Alteration in the Rhythm of the Heart-sounds.—*Reduplication of the Diastolic Sounds.*—This is probably due to a lack of synchronism in the closure of the aortic and pulmonary valves. It is frequently noted in health at the end of a long inspiration. Pathologic reduplication may occur whenever the pressure in either the pulmonary circulation or the peripheral arteries is abnormally increased.

It is a common sign in mitral stenosis, emphysema, arteriosclerosis, and pericarditis.

Embryocardia.—This term is used to indicate a rhythm that resembles that of the fetal heart. The pauses between the sounds are of equal length, and the two sounds are exactly alike. Embryocardia indicates great enfeeblement of the heart and may be observed in low fevers and in cardiac failure from any cause.

Gallop or Cantering Rhythm.—This suggests the hoof-beats of a galloping horse. One of the cardiac sounds is doubled and the diastolic pause is shortened. It indicates extreme weakness of the heart.

Adventitious Sounds.—*Murmurs* are abnormal sounds produced in the heart or blood-vessels. They may result from: (1) Obstruction or regurgitation at the valves in consequence of valvular endocarditis. (2) Dilatation of the ventricle or relaxation of its walls, in consequence of which the auriculoventricular valves become relatively insufficient. (3) Roughening of the valves or of the intima of the great vessels. (4) Aneurysm (bruit). (5) Anemia (hemic murmur).

Exocardial murmurs are adventitious sounds of cardiac origin produced in the pericardium (pericardial friction-sound) or in the pleura or lung, adjacent to the heart (pleuropericardial friction-sound and cardiorespiratory murmur).

Pericardial Friction-sound.—This is an adventitious sound produced in pericarditis by roughening of the serous membrane. It is a harsh, grating, to-and-fro sound, quite superficial, often intensified by pressure with the stethoscope, and generally heard best in the fourth interspace near the sternum. It often varies in intensity from hour to hour, and is rarely transmitted beyond the precordial region.

Pleuropericardial Friction-sound.—This is a sound closely resembling the pericardial friction-sound, but produced by inflammation of that part of the pleura that overlaps the heart. It is intensified by deep inspiration, and often disappears when the breath is held during expiration.

Cardiorespiratory Murmur.—This is a rare adventitious sound, produced by the rhythmic expulsion of air from the lappet of lung covering the heart by the cardiac contractions.

The exact condition under which it occurs is not known. It is usually heard best at the end of inspiration, and is nearly always systolic in time. It is greatly modified by position, deep breathing, coughing, and holding the breath.

Aneurysmal Murmur, or Bruit.—In a certain proportion of cases a murmur is heard in aneurysm. It is systolic in time, heard with greatest intensity over the sac of the aneurysm, and transmitted into the vessels of the neck. There is nothing in the character of the murmur to suggest its origin.

Hemic Murmurs.—Hemic murmurs have the following characteristics: They are soft and blowing in character; they are usually systolic in time; they are usually heard best over the pulmonic area; they are associated with evidences of anemia; they are not accompanied by signs of cardiac enlargement; they are often associated with a continuous hum in the veins of the neck; and they are more affected by deep breathing, position, and exercise than the murmurs of organic disease.

THE PULSE.

The average frequency of the pulse in the healthy adult at rest is between 70 and 80. In new-born infants it is between 130 and 140, and in young children between 90 and 100.

Increased Frequency of the Pulse (Tachycardia).—Habitual frequency is sometimes noted in health. The frequency may be temporarily increased by erect posture, excitement, eating, and the use of stimulants.

Abnormal frequency may result from—(1) Pyrexia. The pulse usually bears a definite relation to the temperature, but in certain diseases, as scarlet fever and septicemia, it may be disproportionately rapid, and in others, like meningitis and yellow fever, it may be disproportionately slow. (2) Exophthalmic goiter. (3) Organic heart disease. (4) Lesions at the base of the brain sufficient to depress the pneumogastrics, as hemorrhage, tumor, and advanced meningitis. (5) Reflex irritation, as in dyspepsia or ovarian or uterine disease. (6) An independent neurosis (essential paroxysmal tachycardia). (7) Action of certain drugs—belladonna, nitrites, thyroid extract, etc. (8) Rheumatoid arthritis (Sansom).

Infrequency of the Pulse (Bradycardia).—*Physiologic slowness* is noted after fasting, sometimes in the puerperium, and habitually in certain persons (50 to 60 a minute).

Pathologic infrequency is observed in many conditions, notably—(1) In certain forms of organic heart disease, especially chronic myocardial disease and aortic stenosis. (2) In jaundice. (3) From pressure at the base of the brain sufficient to irritate the vagus, as in beginning meningitis, tumor, etc. (4) At the close of febrile diseases, as typhoid fever, pneumonia, etc. (5) After the use of certain drugs, as digitalis, aconite, etc.

Heart-block (Adams-Stokes Disease).—This condition is characterized by permanent or paroxysmal bradycardia, cerebral attacks of an epileptiform, vertiginous, or syncopal character, and pulsations of the cervical veins exceeding in rate those of the arteries. It depends upon a lesion of the muscular bundle of His (a narrow band extending from the right auricle and its valves to the interventricular septum), in consequence of which the auricles contract more or less normally, but the ventricles only respond to every other, every second, or every third stimulus (auriculo-ventricular dissociation.)

In young adults Adams-Stokes disease is often of syphilitic origin; in the aged it is usually an expression of myocardial degeneration.

Irregular Rhythm (Arhythmia).—**The Intermittent Pulse.**—This *per se* is not characteristic of any pathologic condition. It is occasionally met with in healthy persons, especially after exercise, mental excitement, or eating. As a pathologic symptom it may result from the excessive use of tea, coffee, or tobacco; from organic disease of the heart, especially myocarditis; reflex irritation, such as flatulent dyspepsia; gout, neurasthenia, or hypochondriasis.

The Bigeminal and Trigeminal Pulses.—These are pulses in which the beats occur in groups of two or three respectively. They are most frequently seen in chronic myocardial disease and in uncompensated mitral stenosis, especially after the too free use of digitalis.

There may be a false intermission in the radial pulse when the heart fails to transmit all its beats to the wrist. This condition may be observed in any disease that weakens the ventricular walls.

The Irregular Pulse.—This is met with in the same conditions as the intermittent pulse. It may be habitual or occasional. There is often marked irregularity in uncompensated mitral regurgitation and chronic myocardial disease. Extreme irregularity with rapidity of the heart-beats is spoken of as *delirium cordis*. It occurs in the late stages of uncompensated heart disease.

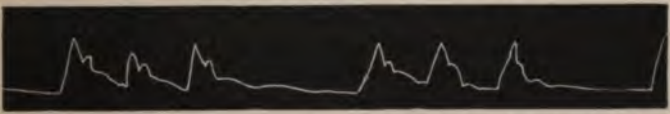

FIG. 8.—Sphygmogram of the trigeminal pulse.

The Pulsus Paradoxus.—This is a pulse in which the wave becomes small and feeble during full inspiration. It is sometimes observed in healthy persons. It is not infrequent in adherent pericardium.

The Dicrotic Pulse.—This is a pulse in which the main beat is quickly followed by a secondary wave or slight rebound of the vessel. It is especially apt to occur when the tension is low and the arteries are relaxed, as in low fevers, like typhoid.

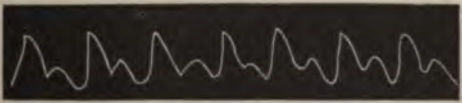

FIG. 9.—Sphygmogram of a dicrotic pulse.

Other Variations in the Pulse.—The High-tension Pulse.—This is a pulse in which the force of the beat is relatively increased. The tension may be roughly estimated by noting the amount of pressure of the fingers that is required to arrest the beat. It may be determined more accurately by means of the sphygmograph.

A *high tension* is observed in many conditions, notably in cardiac hypertrophy; in chronic nephritis, especially interstitial nephritis; in arteriosclerosis; in certain intoxications, like gout, chronic lead-poisoning, and uremia; in cerebral affections irritating the vasometer centers, such as apoplexy and tumor; and in contraction of the arterioles, as in chills and in some paroxysms of angina pectoris.

The Low-tension Pulse.—A low-tension pulse is one that is soft and compressible. It is observed in many conditions, notably in marked cardiac degeneration, in collapse, in low fevers, and in states of great exhaustion and depression.

Venous Pulse.—A true jugular pulsation occurs in tricuspid regurgitation. A venous pulse in the dorsum of the hand may be due to forcible propulsion of the blood through the capillaries, as in compensated aortic regurgitation, or to extreme relaxation of the arterioles, permitting the transmission of the pulse-wave, as in grave anemia.

Capillary Pulse.—This may be detected by the occurrence of systolic flushing in a hyperemic area of the skin (preferably over the forehead) caused by friction, or in the everted lip which has been somewhat blanched by pressure of a glass slide. A capillary pulse is occasionally observed in aortic regurgitation, in grave anemia, and in neurasthenia.

Asymmetric radial pulses may result from: (1) Anomalies in the distribution, size, or division of one of the vessels. (2) Aortic aneurysm. (3) An embolus or an atheromatous plate within the vessel. (4) Fractures, luxations, or inflammatory exudations causing compression of the vessel. (5) Compression of one vessel by tumors within or without the thorax.

"Water-hammer Pulse" (Corrigan's Pulse).—This pulse is characterized by a quick, powerful beat, which suddenly collapses or recedes. The peculiar pulsation may be distinctly visible, not only in the carotids, but throughout the brachial artery. This pulse is diagnostic of aortic regurgitation during the period of compensation, and its force is due to the excessive ventricular hypertrophy and to the large amount of blood expelled with each systole; its sudden recession is due to the incompetent valves failing to support the column of blood in the aorta.

PALPITATION.

Definition.—A rapid and tumultuous action of the heart perceptible to the patient. Rapidity not perceptible to the patient is not termed palpitation.

Etiology.—It may result from: (1) Reflex irritation, as from flatulent distention of the stomach. (2) Excitement, mental or physical. (3) Organic heart disease. (4) Exophthalmic goiter. (5) Overwork, as in the "irritable heart" of untrained recruits. (6) Anemia. (7) Hysteria. (8) An independent neurosis (essential paroxysmal tachycardia).

DROPSY.

Definition.—An unnatural collection of serous fluid in the tissues or cavities of the body.

Etiology.—Dropsy may result from: (1) Chronic visceral affections that bring about venous stasis, as chronic heart disease, cirrhosis of the liver, and emphysema. (2) Local obstruction to the venous circulation by emboli, thrombi, tumors, etc. (3) Changes in the composition of the blood, as in anemia. (4) Increased permeability of the capillary walls, as in Bright's disease. (5) Disturbed innervation, as in hysteria, angioneurotic edema, and neuritis.

GENERAL CYANOSIS.

Definition.—Blueness of the surface from insufficient oxidation of the blood.

Etiology.—Cyanosis results from: (1) Affections preventing the free entrance of air to the lungs, as in laryngeal or tracheal stenosis, asthma, emphysema, pneumonia; (2) obstruction to the venous return, as in uncompensated cardiac disease; (3) congenital heart disease, in which there is venous congestion or a direct admixture of arterial with venous blood; (4) chronic splenomegalic polycythemia (see p. 161); (5) conditions associated with methemoglobinemia, as acetanilid or antipyrin poisoning.

DISEASES OF THE PERICARDIUM.

PERICARDITIS.

Definition.—Inflammation of the pericardium.

Etiology.—(1) It is usually secondary to infectious diseases, such as rheumatism, chorea, the specific fevers, septicemia, and tuberculosis. (2) It may result from the extension of inflammation from the pleura, lung, esophagus, or the heart itself. (3) It may be due to traumatism. It occasionally occurs in Bright's disease. The organisms most commonly found in the exudate are the streptococci, staphylococci, pneumococci, and tubercle bacilli.

Pathology.—In the early stages the membrane is red, sticky, and lusterless. An exudate is soon formed, which may be serofibrinous, fibrinous, or purulent.

In the **serofibrinous form** there is but little inflammatory lymph, the exudate being composed mainly of straw-colored fluid (from a few ounces to two pints or more), which in favorable cases is gradually absorbed.

In the **fibrinous form** serum is scant. The membrane is covered with a butter-like exudate, which subsequently organizes into fibrous tissue and unites more or less firmly the pericardial surfaces. In some cases the development of fibrous pericarditis is insidious and unattended by any acute symptoms. The adhesions offer resistance to the ventricular contractions, and ultimately induce hypertrophy and dilatation of the heart.

In the **purulent form** the sac may contain from a few ounces to two or three pints of pus. Death usually results, but evacuation of the pus is occasionally followed by union of the pericardial surfaces and slow recovery.

In all forms of pericarditis the myocardium is more or less involved.

Symptoms of Serofibrinous Pericarditis.—The chief symptoms are moderate fever, precordial pain, palpitation, dry cough, and dyspnea. The pulse is at first rapid and forcible; later, weak and irregular.

Physical Signs.—The only typical sign of the first stage (dry pericarditis) is a rough, to-and-fro friction rub, usually heard best at the fourth left intercostal space, and not transmitted beyond the precordium. Pericardial effusion is manifested by several definite signs.

Inspection.—The precordium may bulge, especially in children.

Palpation.—A friction fremitus is occasionally felt. The apex-beat is feeble or lost. A pulsation is sometimes felt in the fourth interspace to the left of the mammary line.

Percussion.—There is a large pyramidal area of dulness, with the apex directed upward. A triangular area of dulness in the fifth right interspace or cardiohepatic angle (Rotch's sign) is a trustworthy sign. A dull area is sometimes detected posteriorly in the left infrascapular region.

Auscultation.—The heart-sounds are indistinct and muffled. They are often disproportionately feeble compared with the strength of the pulse.

Purulent Pericarditis.—The symptoms are those of serofibrinous pericarditis plus those of sepsis—irregular fever, chills, sweats, pallor, and marked leukocytosis. Occasionally the precordial tissues are edematous. It may be necessary to aspirate in doubtful cases.

Adherent Pericardium.—The diagnosis cannot always be made during life. The following signs are suggestive: (1) Enlargement of the heart, especially of the right ventricle; (2) systolic retraction in the region of the apex and posteriorly in the region of the eleventh and twelfth ribs (Broadbent's sign); (3) collapse of the cervical veins during diastole; (4) fixation of the apex-beat, so that it does not move with respiration or change in posture; (5) the pulsus paradoxus (see p. 173). With these signs there are often symptoms of heart-failure—dyspnea, cyanosis, edema, and hepatic enlargement.

Diagnosis.—**Acute Endocarditis.**—The endocardial murmur is soft, not harsh; it is systolic or diastolic, not to and fro; it is more distant; it is heard loudest at a valve-point, not at the base of the heart; it is not confined to the precordium, and is not followed by signs of effusion.

Cardiac hypertrophy develops slowly; the impulse is powerful, the apex-beat is displaced downward, and the sounds are loud.

Cardiac Dilatation.—In this condition the area of dulness is not pyramidal in shape; the enlargement is chiefly downward and does not extend beyond the apex-impulse, as in effusion; the impulse is usually visible and undulatory; the line of demarcation between flatness and pulmonary resonance is not so abrupt as in effusion; and the sounds are usually clear and sharp.

Prognosis.—In the dry and serofibrinous forms the prognosis is good under favorable conditions. In the purulent form the outlook is extremely grave. The fibrinous form, though not immediately fatal, is very serious on account of the secondary changes that it induces in the cardiac muscle.

Treatment.—Absolute rest is imperative. Milk is the most suitable diet. Locally, an ice-bag is serviceable. Leeching is beneficial in robust subjects. Blisters are useful when there is great pain. Opium is often necessary to secure rest and to allay pain. If heart-failure occurs, such stimulants as whisky, strychnin, digitalis, and caffein must be employed.

Pericardial Effusion.—When the effusion is serous, absorption may be aided by the application of small blisters, by the administration of diuretics,—infusion of digitalis,—and by the administration of saline purges. Potassium iodid is of doubtful efficacy. Diaphoretics, particularly pilocarpin, should not be used. When pressure symptoms become urgent or the effusion does not yield after a thorough trial to the measures just mentioned, paracentesis should be performed. The most suitable site for the puncture is in the left fifth intercostal space, about an inch or an inch and a half from the edge of the sternum.

In pericarditis with purulent effusion the indications are to incise the sac and to afford the freest possible outlet for the pus. The mortality of incision is about 60 per cent. In adherent pericardium the treatment is that of dilatation.

OTHER AFFECTIONS OF THE PERICARDIUM.

Hydropericardium (dropsy of the pericardium) results from one of the causes of general dropsy, especially heart disease or nephritis. The physical signs are those of serofibrinous pericarditis in the stage of effusion.

Hemopericardium (blood in the pericardium) may result from traumatism, the rupture of an aneurysm, or the rupture of the heart itself. In cancerous and tuberculous pericarditis the serous exudate is often more or less bloody.

Pneumopericardium (air in the pericardium) is very rare. It may result from traumatism or the rupture of a neighboring air-containing organ. Thus it may be produced by pneumopyothorax, a phthisic cavity, or ulceration of the esophagus or stomach.

DISEASES OF THE HEART.

ENDOCARDITIS.

(Valvulitis.)

Definition.—Inflammation of the lining membrane of the heart. The process is usually confined to the valves.

Varieties.—(1) Simple, benign, or verrucose endocarditis. This begins as an acute affection, but usually leads to chronic sclerotic valvular disease. (2) Ulcerative or malignant endocarditis. No sharp line, either clinically or anatomically, can be drawn between this and simple endocarditis. The terms ulcerative and malignant are used to designate a more intense infection. (3) Chronic or sclerotic endocarditis. This may be the continuation of acute endocarditis or it may be chronic from the outset.

Etiology.—*Acute endocarditis* usually results from acute articular rheumatism, one of the infectious fevers, chorea, or septicemia. Gonorrhea, tuberculosis, and Bright's disease are occasional causes. At least 40 per cent. of all cases of acute articular rheumatism are complicated with endocarditis. The young are more liable to be attacked than the old. Sixty-two of 73 fatal cases of chorea collected by Osler showed endocarditis. Of the infectious fevers, scarlatina and pneumonia are most prone to heart complications.

The *ulcerative* type is prone to develop on valves already the seat of chronic inflammation, but it may be primary. It generally follows septicemia, pneumonia, pneumococcus meningitis, gonorrhea, or one of the specific fevers. It is very rarely due to rheumatism or chorea. The micro-organisms most frequently detected in the lesions are the staphylococcus, streptococcus, and the pneumococcus.

Chronic endocarditis may be congenital, may follow an acute attack, or may result directly from chronic rheumatism, gout, alcoholism, syphilis, or chronic nephritis. Severe muscular strain sometimes induces it.

Pathology.—Postnatal endocarditis most commonly involves the valves of the left side of the heart. Prenatal endocarditis most commonly involves the valves of the right side of the heart.

In simple endocarditis the surface of the valve becomes red, swollen, and lusterless. Later, a row of bead-like vegetations (thrombi) appears along the line of maximum contact, which is about 2 mm. from the free margin of the valve. Microscopically, the endothelium beneath the vegetations shows evidence of necrosis, and the adjacent tissue, a round-cell infiltration. The vegetations may be whipped off by the blood-current and carried to distant organs, as the brain, kidney, or spleen; but in the vast majority of cases, if life is preserved, they are transformed, together with the cellular exudate, into fibrous tissue (*chronic endocarditis*), which not only thickens the valves, but, by contracting, so shortens and distorts them that they are rendered in one instance obstructive to the onward flow of blood, and in another incompetent to close the orifice over which they preside. Finally, retrograde changes ensue, the thickened valves becoming fatty and calcareous.

The myocardium is probably more or less involved in every case of endocarditis.

The *ulcerative* type is characterized by more extensive necrosis, the development of ulcers, and the passage into the circulation of septic emboli.

Symptoms of Acute Simple Endocarditis.—Subjective phenomena are often absent, and auscultation may furnish the only indication of endocarditis—namely, a prolongation of the heart-sound, which later develops into a distinct murmur.

In many cases fever, an irregular and rapid pulse, palpitation, precordial distress, and dyspnea are associated symptoms.

Symptoms of Acute Ulcerative Endocarditis.—The **general symptoms** may resemble those of septicemia or typhoid fever. Thus, there may be moderately high and irregular fever, profuse sweats, chills, leukocytosis, delirium, and stupor. Diarrhea is not uncommon.

Cardiac Symptoms.—There may be precordial pain, palpitation, dyspnea, a rapid and irregular pulse, and a murmur at one or other of the valve-points. This murmur is prone to change considerably in intensity and in timbre from day to day. Occasionally there is no murmur or other evidence of cardiac inflammation.

3. **Embolic Symptoms.**—Emboli in the peripheral vessels may occasion a petechial rash; renal embolism may occasion hematuria; splenic embolism, a painful enlargement of the spleen; and cerebral embolism, paralysis.

Diagnosis.—**Typhoid Fever.**—The gradual onset, the more regular fever, the abdominal symptoms, the roseolar rash, the Widal reaction, the bronchial catarrh, the early enlargement of the spleen, and the absence of leukocytosis and of embolic phenomena will serve to separate typhoid fever from ulcerative endocarditis.

Malarial Fever.—This may be recognized by the presence of the malarial parasite in the blood.

Prognosis.—Acute simple endocarditis does not often prove fatal, but it rarely leaves the valve undamaged. Under favorable conditions, however, compensatory hypertrophy of the heart ensues and good health is preserved for an indefinite period. Rapid dilatation of the heart indicates concurrent myocarditis and is a serious sign. Ulcerative endocarditis generally proves fatal in from one to eight weeks. Occasionally the disease lasts several months.

Treatment.—The treatment of acute endocarditis is mainly that of the causal condition. *Prolonged and complete rest* is of the greatest importance. The patient should be confined to bed not only during the attack, but for several weeks after it has subsided, in order to allow sufficient time for the damage to be repaired or for compensatory hypertrophy to be established.

Externally, an ice-bag is often useful in allaying excitement of the heart. Mild mercurial or saline aperients may be used from time to time for their depurative effect. Digitalis may be of service when the pulse is weak and irregular, but generally it is not indicated. Heart-failure is to be combated by such stimulants as alcohol, ammonia, strychnin, and caffein. Repeated vesication and the prolonged use of potassium iodid have been warmly advocated.

CHRONIC VALVULAR DISEASE.

PERIOD OF COMPENSATION.

Compensation is effected by an increase in the strength and size of certain cardiac chambers sufficient to enable the arterial system to receive its normal supply of blood, notwithstanding obstruction or regurgitation at one or more of the valves.

The duration of this period is indefinite, and depends largely on the amount of damage sustained by the heart and the hygienic conditions to which the patient is subjected.

During perfect compensation endocarditis is indicated by physical signs, symptoms being entirely absent.

Aortic Stenosis or Aortic Obstruction.—Definition.— Obstruction to the flow of blood into the aorta from thickening or adhesion of the aortic segments. Uncomplicated aortic stenosis is a rare lesion. It occurs usually in elderly persons.

Physical Signs.—*Inspection.*—If the heart is strong, the apex-beat is forcible and is noted downward and to the left.

Palpation confirms inspection, and often detects a systolic thrill at the base of the heart.

Percussion may yield an increased area of cardiac dulness, especially to the left.

Auscultation.—There is a harsh systolic murmur, heard best in the second right intercostal space, and transmitted in the large vessels of the neck. The aortic second sound is often inaudible or very feeble.

Pulse.—The pulse is apt to be small and infrequent; the wave is long and slow to rise (*pulsus tardus*).

Compensation.—From obstruction to the outflow of blood the left ventricle becomes hypertrophied.

Sequence.—Mitral regurgitation. Weakening and dilatation of the left ventricle prevent perfect closure of the mitral orifice, and relative insufficiency results.

Diagnosis.—The harshness of the murmur, weakness of the second sound, palpable thrill, enlargement of the left ventricle, and especially the characteristic pulse will serve to distinguish this murmur from a basilar systolic murmur caused by anemia or aortic atheroma.

Aortic Insufficiency or Aortic Regurgitation.—**Definition.**—Failure of the aortic valves to prevent a return of blood to the ventricle, from rupture or inflammatory contraction of the segments, or from dilatation of the orifice. It is most frequently seen in middle-aged men, especially in those who have done hard manual work.

Physical Signs.—*Inspection.*—The apex-beat is forcible, and displaced downward and to the left. There is often bulging of the precordium.

Palpation.—This confirms inspection.

Percussion.—There is a marked increase in the area of cardiac dulness, especially toward the left and downward.

Auscultation.—There is a diastolic murmur, heard most distinctly in the second right intercostal space, and transmitted down the sternum and toward the apex. A presystolic murmur (Flint murmur) is sometimes heard at the apex. It is probably due to the impact of the regurgitant stream upon the anterior mitral leaflet.

Pulse.—The arteries, especially the carotids, brachials, and radials, pulsate visibly. Palpation detects the "water-hammer" or Corrigan's pulse—*i. e.*, a short, full, and receding pulse.

The extreme cardiac enlargement makes the pulse full, and the prompt leakage back into the ventricle makes it short and receding. Elevation of the arm during palpation of the radial artery makes this pulse more apparent, as the position favors regurgitation. A capillary pulse is sometimes present. It may be noted at the root of the finger-nail by an alternate blushing and paling, synchronous with the heart-beats.

Compensation.—Dilatation and hypertrophy of the left ventricle. Dilatation results from the reception of such a large quantity of blood during diastole, and hypertrophy follows from the increased effort which the ventricle must put forth in emptying itself of this extra quantity of blood.

This extremely dilated and hypertrophied heart has been called the *cor bovinum*, or ox-heart.

Sequence.—Mitral regurgitation. The dilatation and weakening of the ventricle prevent perfect closure of the mitral orifice, and relative insufficiency results.

Mitral Stenosis or Mitral Obstruction.—**Definition.**—Obstruction to the flow of blood through the mitral orifice, from thickening or adhesion of the mitral segments.

It is usually seen in early life, and is more common in females than in males.

Physical Signs.—*Inspection.*—The apex-beat is not much displaced. There is sometimes bulging over the lower part of the sternum.

Palpation.—There is a rough presystolic thrill near the apex.

Percussion.—The area of dulness is increased to the right of the sternum.

Auscultation.—A presystolic murmur is heard a little within the apex, and is not transmitted. This murmur is prolonged, rough, and churning in character, increases in loudness as it approaches the first sound, and ends in an abrupt systolic shock. The pulmonic second sound is accentuated.

Pulse.—During the period of compensation the pulse is small and regular. After failure of compensation there is often extreme irregularity, both in force and in rhythm.

Compensation.—From obstruction to the outflow of blood the left auricle becomes enlarged; when it loses power, the blood accumulates in the lung, and to overcome this pulmonary resistance the right ventricle becomes hypertrophied.

There is no strain on the left ventricle, and hence that chamber is not enlarged.

Sequence.—Tricuspid regurgitation. Dilatation of the right ventricle prevents perfect closure of the tricuspid orifice, and relative insufficiency results.

Diagnosis.—The loud systolic shock, accentuation of the pulmonic second sound, and enlargement of the right ventricle will serve to distinguish this murmur from the *Flint murmur* (see p. 183).

Mitral Insufficiency or Mitral Regurgitation.—**Definition.**—Imperfect closure of the mitral orifice from rupture or inflammatory contraction of the mitral segments; or from dilatation or weakening of the left ventricle, preventing perfect coaptation of normal valves. Mitral regurgitation is the most frequent of the valvular defects.

Physical Signs.—*Inspection.*—The apex is usually to the left and downward. There may be bulging of the precordium. *Palpation* confirms inspection.

Percussion.—The area of dulness is increased transversely, especially toward the right.

Auscultation.—The murmur is systolic, loudest at the apex, and transmitted to the left axilla and angle of the scapula. The pulmonic second sound is accentuated.

Pulse.—During the period of compensation the pulse may be full and regular. It usually becomes quite irregular when the heart weakens.

Compensation.—The left auricle enlarges from the extra amount of blood that it receives; when it weakens, the lungs become congested and right ventricular hypertrophy follows.

The left ventricle also becomes hypertrophied, from its effort to move the large quantity of blood which it receives from the distended auricle during each diastole.

Sequence.—Tricuspid regurgitation. Weakening and dilatation of the right ventricle prevent perfect closure of the tricuspid orifice.

Tricuspid Stenosis or Tricuspid Obstruction.—This lesion is extremely rare, and difficult to distinguish from mitral stenosis, with which it is usually associated. It gives rise to a transverse enlargement of the heart and a presystolic murmur, which is heard loudest over the base of the ensiform cartilage.

Tricuspid Insufficiency or Tricuspid Regurgitation.—**Definition.**—Imperfect closure of the tricuspid orifice from inflammatory shortening of the valves; or, more commonly, from dilatation of the right ventricle secondary to mitral disease or to chronic lung disease.

Physical Signs.—The characteristic signs are enlargement of the heart, especially to the right of the sternum; a systolic murmur, loudest over the lower portion of the sternum; a systolic pulse in the jugular veins and in the liver.

Pulmonary Stenosis or Pulmonary Obstruction.—This very rare lesion is always congenital. It may be suspected when there are marked enlargement of the right ventricle, a systolic murmur in the second left intercostal space which is not transmitted into the vessels of the neck, a systolic thrill in the same area, and persistent cyanosis.

Pulmonary Insufficiency or Pulmonary Regurgitation.—This is very rare, and is usually congenital. It produces a diastolic murmur in the second left intercostal space.

PERIOD OF BROKEN COMPENSATION.

Broken compensation usually results from: (1) Increasing damage to the valves; (2) senility, leading to arterial and cardiac degeneration; (3) some intercurrent disease, throwing additional strain on the heart; or (4) undue physical exertion.

During this period subjective symptoms appear. In cardiac insufficiency, no matter what the original valvular lesion may have been, the heart becomes unable to fill the arteries, and the blood is dammed back in the lungs, and venous congestion of the organs follows.

Symptoms.—Pulmonary congestion produces dyspnea, hemoptysis, and often chronic bronchial catarrh with cough and expectoration.

Hepatic, stomachic, and intestinal congestion produces dyspepsia. Renal congestion produces scanty, albuminous urine, and later nephritis.

General venous congestion produces cyanosis and dropsy. The latter is first noted in the feet.

Disturbances of the cerebral circulation produce headache, vertigo, and syncopal attacks.

In aortic disease, especially aortic stenosis, cerebral symptoms are often marked. In mitral disease pulmonary symptoms are usually marked.

Prognosis of Chronic Valvular Affections.—The extent of damage can never be accurately determined by the quality or intensity of the murmur.

All things being equal, the following is probably the order of gravity of the various valvular lesions: (1) Tricuspid regurgitation; (2) aortic regurgitation; (3) mitral stenosis; (4) aortic stenosis; and (5) mitral regurgitation. Sudden death is rare, except in aortic regurgitation.

The following conditions are unfavorable: Early childhood, advanced years, distinct liability to recurring attacks of rheumatism, great cardiac enlargement, irregular heart-action, symptoms of pulmonary, gastric, and renal congestion, and poor hygienic surroundings.

In proportion to the absence of these conditions the prognosis becomes favorable. In many cases life is not materially shortened.

Treatment.—When compensation is well maintained, the treatment should be purely hygienic.

Stage of Broken Compensation.—Absolute rest is the most important element in the treatment. The diet should be nutritious but readily digestible. In some cases it may be well to restrict the diet for a time to milk, giving 3 or 4 ounces every two hours. The most reliable cardiac stimulant is digitalis (10 to 20 minims of the tincture two or three times a day). It may be given in any form of valvular disease when there are dyspnea, edema, deficient urination, and a frequent, weak, irregular pulse. Strophanthus, spartein, and caffein sometimes succeed when digitalis fails. Strychnin is the most valuable adjuvant to digitalis, especially when there are degenerative changes in the heart. Mercurial and saline aperients are useful in lowering venous tension, and without their aid digitalis may fail. When the right ventricle is greatly overdistended and cyanosis is marked, venesection to the extent of a pint or more may prove life-saving. Iron and arsenic are very serviceable when there is anemia. They may sometimes be combined advantageously with digitalis and strychnin, as in the following pill:

℞.	Arseni trioxidi	gr. $\frac{1}{3}$
	Massæ ferri carbonatis	gr. xx
	Strychninæ sulphatis	gr. ss
	Pulveris digitalis	gr. x–xx.—M.

Fiant pilulæ No. xx.
SIG.—One pill after meals.

Special Symptoms.—Dropsy.—The most useful measures are hydragogue cathartics (salines in concentrated solution; compound jalap powder, 30 to 40 grains; and elaterium, $\frac{1}{8}$ grain); diuretics (digitalis, caffein, vegetable salts of potassium); the application of smooth, firm bandages to the limbs; the introduction of fine silver cannulæ (Southey's tubes), and incisions behind the ankles.

Dyspnea may yield to cupping, sinapisms, the administration of Hoffmann's anodyne, and, in cases of high arterial tension, to nitrites. Morphin is especially useful in relieving nocturnal dyspnea.

Restlessness and Insomnia.—On the whole, morphin ($\frac{1}{6}$ grain) with atropin ($\frac{1}{150}$ grain) is the best sedative. Trional, bromids, and chloralamid are worthy of confidence.

Pain.—Temporary oppression is often relieved by warm or cold applications and the administration of Hoffmann's anodyne. Severe continuous pain may yield to leeching or blistering. In anginoid pains nitrites and potassium iodid are often efficacious.

Sudden heart-failure must be met by the administration of diffusible stimulants, such as ammonia, whisky, and ether. The application of heat to the precordium is useful.

ENLARGEMENT OF THE HEART.

Varieties.—(1) *Simple Hypertrophy.*—The muscle of the heart is increased in thickness, but the cavities are of normal size.

(2) *Eccentric Hypertrophy* (*Hypertrophy with Dilatation*).—The muscle is thickened and the cavities are increased in size.

(3) *Simple Dilatation.*—The muscle is thinned and the cavities are increased in size.

Etiology.—*Hypertrophy* is always the result of increased demands upon the functions of the heart. Thus, it may be due to—(1) Valvular disease; (2) abnormal resistance in the peripheral circulation, as in arteriosclerosis and chronic Bright's disease (left ventricle); (3) abnormal resistance in the pulmonary circulation, as in emphysema and cirrhosis of the lung (right ventricle); (4) prolonged exertion, as in athletes; (5) long-continued palpitation or tachycardia, as in exophthalmic goiter or tobacco heart; (6) interference with the ventricular contractions by pericardial adhesions.

Dilatation of the heart results from the same causes. It is more apt to occur than hypertrophy when the demands are sudden and severe, or when they fall upon a heart the walls of which are already degenerated.

Pathology.—In *hypertrophy* the muscle of the heart is firm and of a dark-red color. The normal weight (8 or 9 ounces) may be doubled or trebled. When the left ventricle is chiefly involved, the organ is increased in length. When the right ventricle is chiefly involved, the organ becomes more globular. Microscopically, the fibers are increased in size and in number.

In *dilatation* the heart muscle is softer, more flabby, and often lighter in color from degenerative changes.

Symptoms.—*Hypertrophy.*—Unless the hypertrophy is more than compensatory, no symptoms result. Excessive hypertrophy may give rise to precordial distress and symptoms of cerebral hyperemia—headache, tinnitus aurium, flashes of light, etc.—and the following physical signs: bulging of the precordium, a heaving impulse, displacement of the apex-beat downward and to the left, an increase in the area of cardiac dulness, a loud, booming first sound, accentuation of the aortic second sound or of the pulmonic second sound, according as the hypertrophy involves the left or right ventricle, and a strong, full pulse.

Dilatation also gives signs of cardiac enlargement, but the impulse is feeble or imperceptible, the first sound is short and weak (clicking), the pulse is rapid and feeble, and often irregular or intermittent, and usually there are symptoms of venous congestion—dyspnea, cough, edema, flatulent dyspepsia, and deficient urination. Soft systolic murmurs, the result of relative mitral or tricuspid insufficiency, may be heard.

Treatment.—In *hypertrophy* treatment is rarely called for. Mercurial and saline aperients are useful in lowering arterial tension. Aconite may be used cautiously. The cerebral symptoms will generally yield to bromids or to vasodilators, like the nitrites.

The treatment of dilatation is for the most part that of valvular disease in the stage of broken compensation. Digitalis, strophanthus, and strychnin are the most reliable remedies in chronic cases. In acute cases diffusible stimulants—alcohol, ether, ammonia—will be required. When the right heart is especially embarrassed and there is orthopnea with cyanosis, venesection is often of the greatest value.

ACUTE MYOCARDITIS.

Definition.—Acute inflammation of the heart muscle.

Etiology.—It results from the same causes as acute endocarditis.

Pathology.—It is usually associated with endocarditis or pericarditis. Occasionally the myocardium is the only part of the heart affected. The inflammatory process is always accompanied with more or less parenchymatous or fatty changes in the muscle-fibers. The essential lesion is the infiltration of the interstitial tissue with round cells.

Symptoms.—The symptoms are often masked by the primary disease. Dyspnea, precordial distress, palpitation, pallor, a feeble impulse, a rapid, weak, irregular, and unstable pulse, and muffling of the heart-sounds may suggest the condition. The heart may or may not be dilated.

Treatment.—The treatment is that of acute endocarditis. Cardiac stimulants—strychnin, alcohol, ammonia, camphor, and caffein—are indicated.

CHRONIC MYOCARDIAL DISEASE.

The chief chronic affections of the myocardium are fatty infiltration, fatty degeneration, and fibroid induration (chronic interstitial myocarditis).

Fatty Infiltration.—This consists in an excess of the fat which is normally present in variable amounts beneath the epicardium, especially along the blood-vessels and in the grooves. In advanced cases the fat is also found between the muscle-fibers. The latter may remain normal for a long period, but ultimately, owing to compression, they undergo atrophy and fatty degeneration. Fatty infiltration results from the causes which lead to general obesity. Thus, it may be induced by an hereditary tendency, by overeating and drinking, and by sedentary habits.

Fatty Degeneration.—This is a degeneration of the muscle-fibers themselves, with the formation of fat. It is frequently due to local anemia from sclerosis of the coronary arteries; it follows hypertrophy in valvular disease; it is a common result of malnutrition from old age, wasting diseases, or grave anemia; it is associated with parenchymatous degeneration (cloudy swelling) in severe infections; it occurs also in acute mineral poisoning, as by phosphorus, antimony, or arsenic.

The muscle of the heart is pale, soft, and flabby, and feels greasy to the touch. Microscopically, the fibers are found filled with small, dark fat-granules.

Fibroid Induration.—This is met with as a replacement fibrosis secondary to sclerosis of the coronary arteries. It may be secondary, however, to chronic endocarditis or pericarditis. The indirect causes are old age, gout, alcoholism, syphilis, and rheumatism.

Symptoms of Fatty Infiltration.—In fatty infiltration symptoms are not marked unless the muscle-fibers themselves are affected. It may be suspected in an obese subject, when the only symptoms present are dyspnea on exertion, palpitation, weak heart-sounds, and a feeble but regular pulse.

Symptoms of Fatty Degeneration and of Fibroid Induration.—These two conditions cannot be differentiated clinically. The symptoms are very variable. There is often a *sense of oppression or discomfort* in the region of the heart. Attacks of *angina pectoris* may occur. *Dyspnea* is rarely absent. Toward the end *Cheyne-Stokes breathing* may develop. The *pulse is usually weak and irregular*, both in force and in rhythm; sometimes it is intermittent, and occasionally it is extremely slow (40 or 50 a minute). The *first sound of the heart is feeble and muffled;* the second aortic sound is often relatively accentuated. The heart is not necessarily enlarged. There may be no murmurs, but a systolic murmur is often heard at the apex in consequence of relaxation of the mitral sphincter. There may be *edema* of the feet, and even anasarca. *Progressive weakness* and *pallor* often develop from increased venous tension and interference with absorption. Attacks of *asystole* (orthopnea, cyanosis, pulmonary edema, and delirium cordis) are occasionally observed. In rare instances the *Adams-Stokes syndrome* (infrequency of pulse with syncopal, epileptiform, or vertiginous attacks) is present.

The *history*, *age of the patient*, and *condition of the arteries* must also be considered in making the diagnosis.

Prognosis.—Serious. Sudden death may occur at any time.

Treatment of Fatty and Fibroid Heart.—Laborious work, mental strain, and excitement should be avoided, as far as possible. The diet should be simple and easily digestible. When the pathologic changes are not far advanced, and particularly if they consist in fatty overgrowth rather than in degeneration of the muscle-fibers, graduated exercise, coupled with warm saline baths, as in the well-known Nauheim treatment, may be very efficacious. Constipation must be relieved.

As to special treatment, strychnin ($\frac{1}{60}$ to $\frac{1}{30}$ grain thrice daily) is the most generally useful drug. When there is anemia, iron and arsenic are excellent adjuvants. Nitrites are beneficial in cases with high arterial tension, anginoid pains, or cardiac asthma. Digitalis is serviceable in some cases; it should be given cautiously, however, and in small doses.

ANGINA PECTORIS.

(**Neuralgia of the Heart; Stenocardia.**)

Definition.—A symptomatic affection most commonly associated with occlusion of the coronary arteries and degeneration of the myocardium, and characterized by severe paroxysmal pain in the region of the heart and a sense of imminent death.

Etiology.—It usually develops after middle life, and is very much more common in men than in women. The predisposing causes are those of arteriosclerosis—*i. e.*, alcoholism, gout, and syphilis. In some instances an hereditary tendency has been noted, and not infrequently the attacks have been preceded by prolonged mental anxiety.

A false angina (*pseudo-angina pectoris*) is sometimes associated with hysteria, or the excessive use of tobacco.

Pathology.—Obstruction of the coronary arteries from atheroma, thrombosis, or embolism, with resultant degeneration of the myocardium, is the condition usually found after death. Occasionally, typical attacks occur in lesions of the aortic valve, especially insufficiency, and in aortic aneurysm.

Symptoms.—The attacks are usually excited by strong emotion, muscular effort, or flatulent indigestion, and are characterized by agonizing pain, radiating from the heart to the shoulder and arm (usually the left), a sense of impending death, immobility of the body, dyspnea, and a pale, anxious face. The pulse is very variable. The attacks last from a few seconds to several minutes. Death may occur in the first attack, or there may be recurring attacks over a period of many years.

Hysteric Angina.—This neurosis is seen chiefly in women, whereas true angina is very rare in women; there is no evidence of organic heart disease; the attack is longer in duration; there is no immobility of the body; emotional outbreaks, such as moaning and crying, are common, and vasomotor phenomena are often pronounced.

Gastralgia.—The pain is apt to appear when the stomach is empty, and is relieved by stimulating food; it does not radiate to the shoulder and arm; there is no sense of impending death, no fixation of the body, and no evidence of structural heart disease.

Prognosis.—Grave. Sudden death may occur at any time. In false angina the prognosis is favorable.

Treatment.—The general treatment is that of chronic myocardial disease. The most valuable special remedies, in the order of their efficacy, are the nitrites, iodids, and arsenic.

The Attack.—No drug is so generally useful as amyl nitrite (3 to 5 minims on a handkerchief). Marked flatulency should be met by the prompt administration of Hoffmann's anodyne or spirit of mint. When the attacks are severe and prolonged, morphin (¼ to ⅓ grain) should be given hypodermically. When these remedies fail, recourse should be had to chloroform inhalations. The application of heat to the precordium is useful. Cardiac depression following the seizures should be combated by strychnin, ammonia, camphor, or ether.

DISEASES OF THE ARTERIES.

ANEURYSM OF THE AORTA.

Definition.—A more or less localized dilatation of the aorta.

Etiology.—The predisposing causes are those of arteriosclerosis—syphilis, alcoholism, gout, rheumatism, lead-poisoning, and nephritis. Of these, syphilis is by far the most potent factor. Immoderate physical exertion is the most common exciting cause. More than 80 per cent. of all cases occur in males. It is most frequent between the ages of thirty and fifty.

Pathology.—Aneurysms are divided, according to shape, into *fusiform*, *saccular*, and *cylindric* forms. Rupture of the intima, with the passage of blood between the outer tunics, constitutes a *dissecting aneurysm*. A *false aneurysm* is one in which all the tunics are ruptured and the extravasated blood is circumscribed by the surrounding tissues.

The adventitia and intima are much thickened, while the media is thinned or deficient. Fibrinous deposits are usually found in the interior of the sac, especially in the saccular and fusiform varieties.

The arch of the aorta is the most common seat. About 5 per cent. of aortic aneurysms are abdominal.

THORACIC ANEURYSM.

Physical Signs.—*Inspection* may reveal a circumscribed bulging and an abnormal area of pulsation. Dilatation of the superficial veins may also be noted, and in advanced cases the skin over the prominence may be red and glossy.

Palpation.—This may detect an expansile pulsation, a systolic thrill, and a diastolic shock from the recoil of the blood in the sac.

In aneurysm of the transverse arch a downward tug of the trachea is sometimes felt when the head is thrown back and the cricoid cartilage is grasped between the fingers and thumb.

Percussion may reveal an abnormal area of dulness with increased resistance.

Auscultation.—Unless the sac contains too much fibrin, the ear may detect marked accentuation of the diastolic sound and a systolic murmur or bruit.

Radioscopy may demonstrate a shadow corresponding to the location of the aneurysmal sac.

Pulse.—There may be inequality of the radial pulses, owing to partial blocking of a main arterial branch or to pressure on the innominate or one of the subclavian arteries by the sac itself.

Pressure Effects.—*Dyspnea* with *stridulous inspiration* may result from pressure on the trachea or a bronchus. *Parox-*

ysmal croupy cough may be excited by pressure on the trachea or recurrent laryngeal nerve. *Aphonia* may also result from pressure on the recurrent laryngeal nerve. *Dysphagia* may result from pressure on the esophagus. *Pain* of a boring or lancinating character may arise from pressure on neighboring nerve-trunks or bones. *Dilatation or contraction of one pupil* and *unilateral sweating* may be excited by pressure on the sympathetic.

Edema and cyanosis of the one arm and shoulder may follow pressure on the large venous trunks.

Diagnosis.—**Mediastinal tumor** may simulate aneurysm, but in the former the pulsation is not expansile, there is no diastolic shock, the tracheal tug is usually absent, and there may be cachexia, enlargement of superficial glands, and leukocytosis.

Pulsating Empyema.—A left-sided purulent effusion may transmit a cardiac pulsation, but there is no diastolic shock, no thrill, and no murmur. The history, moreover, will usually suggest pleurisy.

Aortic Stenosis.—In this condition there are no evidences of a tumor, no pressure symptoms, and no changes in the pulses.

Prognosis.—Grave. Death usually occurs in from one to two years from rupture, asphyxia, exhaustion, cerebral embolism, or inflammation of a lung ("aneurysmal phthisis"). Rupture may take place into the trachea, a bronchus, the esophagus, lung, pleura, or pericardium, or externally. In rare instances recovery follows from clot-formation.

Treatment.—The treatment commonly employed is a modification of Tufnell's method, and consists in absolute rest in bed for a period of six or eight weeks, a comparatively dry diet, and the administration of potassium iodid (10 to 20 grains thrice daily). For severe pain the most effective measures are the application of an ice-bag and the administration of nitroglycerin or morphin. When there is marked dyspnea with cyanosis, venesection may afford prompt relief. Attempts have been made to favor coagulation by injecting gelatin (100 c.c. of a 5 per cent. sterilized solution) subcutaneously, or by inserting into the sac fine gold wire and passing through the wire a strong galvanic current (Moore-Corradi treatment).

ANEURYSM OF THE ABDOMINAL AORTA.

Seat.—It is most frequently located near the celiac axis.

Symptoms.—It may be recognized by sharp pain in the back, radiating along the spinal nerves, by a delay in the femoral pulse, by gastro-intestinal symptoms, and by physical signs similar to those of thoracic aneurysm.

Diagnosis.—An **abdominal tumor** may receive a pulsation from the aorta and simulate aneurysm, but in the former the pulsation is not expansile, and is frequently lost when the patient is placed in the knee-breast posture.

Expansile Abdominal Aorta.—This is most frequently seen in women, in whom abdominal aneurysm is very rare; the pulsation is often paroxysmal; there is no distinct tumor, and there are no pressure symptoms.

Prognosis.—Very grave. Death usually results from rupture. Occasionally the fatal issue is effected through erosion of the vertebræ and paraplegia, or through embolism of the superior mesenteric artery.

Treatment.—Same as in thoracic aneurysm.

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ARTERIOSCLEROSIS.

(Atheroma; Chronic Endarteritis.)

Definition.—A circumscribed or diffuse thickening of the arterial walls, especially of the intima, secondary to certain degenerative changes in the media.

Etiology.—It is a natural accompaniment of old age. The causes that favor its early development are alcoholism, syphilis, gout, Bright's disease, rheumatism, chronic lead-poisoning, and excessive muscular strain.

Pathology.—The arteries are thickened, tortuous, and rigid. The intima of the large vessels reveals roughened and opaque areas that may be the seat of calcareous deposits. In extreme cases there may be spots of necrotic softening in the subendothelial tissue, forming so-called "atheromatous abscesses."

Microscopically, the muscular fibers of the media are atrophied and the seat of fatty degeneration or calcification. In the intima there is marked hyperplasia of the subendothelial connective tissue, the cells of which may be the seat of hyaline, fatty, or calcareous degeneration. The adventitia is also the seat of connective-tissue overgrowth.

Symptoms.—These vary with extent and distribution of the sclerosis. When the process is general, it may be recognized by rigidity of the peripheral vessels, a sluggish, high-tension pulse, accentuation of the second aortic sound, and enlargement of the left ventricle.

When the *coronary arteries* are especially involved, the symptoms of chronic myocardial disease appear. When the *renal vessels* are especially affected, there may be symptoms of chronic interstitial nephritis. Involvement of the *cerebral arteries* may be indicated by headache, vertigo, insomnia, mental sluggishness, and, perhaps, transient paralysis.

Sequels.—Cerebral hemorrhage or thrombosis, chronic myocardial disease, angina pectoris, interstitial nephritis, aneurysm, and gangrene of the extremities.

Treatment.—Treatment should be directed to the underlying diathesis. Alcohol should be forbidden. Overexertion, both mental and physical, is injurious. Gentle exercise in the open air, however, may be recommended. Heavy feeding must be restricted. The periodic use of mild mercurial or saline aperients is very beneficial. Potassium iodid and the nitrites are often useful when the blood-pressure becomes too high.

DISEASES OF THE RESPIRATORY SYSTEM.

THE NOSE.

Movement of the Alæ Nasi during Respiration.—Playing of the alæ is occasionally noted in health, but it is generally an indication of some obstruction to the entrance of air. It is frequently observed in spasmodic croup, true croup, laryngeal edema, capillary bronchitis, and pneumonia.

Nasal Discharge.—Temporary "running from the nose" is a symptom of acute coryza, measles, hay-fever, diphtheria, and influenza. An offensive discharge should suggest nasal diphtheria or the impaction of a foreign body.

Chronic discharge occurs in chronic rhinitis. In infants, chronic nasal discharge with mouth-breathing ("snuffles") is very suggestive of hereditary syphilis.

The Sense of Smell.—This is tested by holding odoriferous substances before one nostril at a time while the other is closed. Pungent vapors should be avoided, as the irritation which they excite, and not their odor, may lead to their recognition.

The sense of smell may be impaired or lost (anosmia) from:

1. Rhinitis or morbid growths.
2. Affections of the anterior part of the brain, involving the olfactory nerves or bulbs—as injury, tumor, meningitis.
3. Lesions of the olfactory centers (temporosphenoidal lobe).

Paralysis of the trigeminal nerve (by inducing dryness of the mucous membrane).

Extreme acuteness of the sense of smell (hyperosmia) and *perversions of the sense of smell* (parosmia) are sometimes observed in neurasthenia, hysteria, and insanity.

Epistaxis.—Hemorrhage from the nose occurs under the following conditions: (1) Traumatism; (2) inflammation or ulceration; (3) new growths; (4) cerebral congestion; (5) engorgement from chronic heart or liver disease; (6) blood dyscrasia, as in hemophilia, purpura, scurvy, pernicious anemia, etc.; (7) onset of fevers, especially typhoid; (8) in rarefied atmosphere, as in mountain-climbing; (9) vicarious menstruation (rare).

THE LARYNX.

Spasm of the laryngeal adductors is characterized by intense dyspnea and occurs in spasmodic croup; in true croup; in ulceration of the larynx; in laryngismus stridulus; in whooping-cough; in tetany; in hysteria; in hydrophobia; in the laryngeal crisis of locomotor ataxia; when foreign bodies have lodged in the larynx; and when aneurysms or mediastinal tumors press on the recurrent laryngeal nerve and irritate it.

Aphonia or **loss of voice** may be due to: (1) Organic disease of the larynx—inflammation, neoplasms, cicatricial stenosis. (2) Centric paralysis of the recurrent laryngeal nerves, as in bulbar palsy. (3) Peripheral paralysis of the recurrent laryngeal nerves caused by pressure of an aneurysm, mediastinal tumor, or pericardial effusion. (4) Hysteria. (5) The lodgement of foreign bodies. (6) Prolonged use of the voice.

RESPIRATION.

Dyspnea.—Dyspnea implies difficult breathing, with or without an increase in the number of respirations. Dyspnea which is so severe as to necessitate a sitting posture is termed orthopnea. Dyspnea may occur on inspiration, expiration, or both.

Its *chief causes* are: (1) Obstruction in the larynx from spasm, paralysis, false membrane, edema, or a foreign body. (2) Pressure of an aneurysm, tumor, or large glands upon the trachea, a bronchus, or the recurrent laryngeal nerve. (3) Asthma. (4) Diseases of the lungs, as pneumonia, emphysema, edema, phthisis, abscess, and gangrene. (5) Pleural effusions. (6) Cardiac disease. (7) Paralysis of the muscles of respiration. (8) Abdominal distention. (9) Anemia.

Inspiratory dyspnea is frequently seen with tumors or foreign bodies in the larynx.

Expiratory dyspnea is noted in emphysema and occasionally in movable tumors situated below the glottis. In asthma, also, the dyspnea may be largely inspiratory.

The Number of Respirations a Minute.—In the healthy male adult the number of respirations is about 18 a minute. In women and children breathing is somewhat more rapid. The ratio between respirations and pulse-beats is as 1 is to 4 or 4.5.

Rapid respirations are noted in excitement; in pyrexia; in inflammatory diseases of the lungs; in anemia; in certain affections involving the base of the brain; in poisoning from certain drugs that affect the respiratory center; in hysteria; in painful affections of the respiratory muscles, as pleurodynia and pleurisy.

Infrequent respirations are observed in certain diseases of the brain, as meningitis, tumor, apoplexy; in advanced fatty degeneration of the heart; in certain forms of coma, particularly uremic and diabetic; in poisoning with certain drugs, especially opium; in obstruction to the air-passages, as in asthma and in laryngeal spasm.

Cheyne-Stokes, or Tidal-wave Breathing.—In this type the respirations gradually increase in rapidity and volume until they reach a climax, then gradually subside, and finally cease entirely for from five to fifty seconds, when they begin again. It depends on some disturbance of the respiratory center the exact nature of which is still undetermined. It is usually a forerunner of death, but cases have been reported in which it has lasted several months.

Its *chief causes* are: (1) Certain cerebral diseases, as apoplexy, meningitis, and tumor. (2) Advanced cardiac disease, especially fatty degeneration. (3) Certain forms of coma, especially that produced by uremia, opium-poisoning, and sunstroke.

COUGH.

Cough may be induced by: (1) Most organic diseases of the pharynx, larynx, bronchi, and lungs. (2) Foreign bodies in the air-passages. (3) Certain infections which are associated with catarrh, such as typhoid fever, measles, whooping-cough, and influenza. (4) Inhalation of irritant dusts or vapors. (5) Reflex irritation, as from pressure on the recurrent laryngeal nerve or from disease of the abdominal organs. (6) Hysteria.

Laryngeal Cough.—This cough has a hard, metallic, ringing intonation, and has been termed "croupy." It is observed in laryngitis; in whooping-cough; in tuberculosis and syphilis of the larynx; when a foreign body is lodged in the larynx; when the recurrent laryngeal nerve is irritated by pressure of a tumor or aneurysm; and in hysteria.

Dry Cough.—Cough without expectoration is especially observed in the beginning of inflammatory diseases of the bronchi and lungs; in pleurisy; in most chest diseases of early childhood; and in reflex irritation of the larynx.

Moist or loose cough occurs especially in bronchitis, bronchiectasis, abscess of the lung, convalescent pneumonia, and phthisis.

EXPECTORATION.

Mucoid sputum is noted especially in the beginning of acute bronchitis; in asthma; in the early stage of pneumonia and phthisis; and in pulmonary edema. In edema the sputum is very frothy and watery.

Mucopurulent Sputum.—This is observed in subacute and chronic catarrhal affections of the lungs and bronchi, especially in subacute and chronic bronchitis, convalescent pneumonia, and phthisis.

Purulent Sputum.—Sputum is rarely composed of pure pus. Expectoration almost entirely purulent is sometimes observed in bronchiectasis, in phthisis with cavities, in abscess of the lung, and when an empyema ruptures into the lung.

Prune-juice sputum is tinged with altered blood so as to resemble prune-juice. It results from retention of blood in the lung, and is observed in advanced croupous pneumonia, especially low forms, in gangrene of the lung, and in cancer of the lung.

Rusty Sputum.—A rusty and tenacious sputum is strongly indicative of croupous pneumonia.

Currant-jelly sputum is more or less characteristic of cancer of the lung.

Reddish-brown sputum (resembling anchovy sauce) containing amebæ is sometimes observed in hepatopulmonary abscesses.

Sputum containing fibrous shreds is observed in membranous croup, in diphtheria, and in fibrinous bronchitis.

Fetid sputum usually results from bronchiectasis, advanced phthisis with cavities, gangrene of the lung, and abscess of the lung.

Such sputum, when allowed to stand in a conic glass, settles in three layers: an upper layer of dirty froth, a middle layer of turbid mucus in which are suspended purulent strings, and a bottom layer of decomposed pus.

Nummular sputum is sputum occurring in round, flat, coin-shaped masses, which sink in water. Expectoration of this character is seen in advanced phthisis and occasionally in bronchiectasis.

The Microscopy of Sputum.—**Elastic fibers** are found in the sputum in phthisis, abscess, gangrene of the lungs, and in some cases of bronchiectasis.

The Detection of Elastic Fibers.—Place the sputum which has collected during the night in a glass beaker, and add to it an equal volume of a solution of caustic soda (20 grains to the ounce), and boil over a spirit-lamp, stirring it occasionally with a glass rod. As soon as it boils, pour into a conic glass, and add four or five times the amount of cold distilled water. Allow the mixture to stand for two to three hours, and examine the sediment as for tube-casts (Fenwick).

Spirals of Mucin.—Tightly coiled spirals of mucin, which probably represent molds of the fine bronchioles, were first pointed out by Curschmann in the sputum of asthma.

They have also been observed in the sputum of croupous pneumonia.

Charcot-Leyden Crystals.—These are small transparent octahedral crystals, similar to those found in the blood in leukemia. They are observed especially in the sputum of asthma. They have also been noted in phthisis, in fibrinous bronchitis, and in acute bronchitis.

Crystals of Fatty Acids.—These occur as fine needles, singly or in bundles, and are often sharply curved near their extremities. They are observed in the sputum of chronic bronchitis, of abscess, and of gangrene of the lungs.

Crystals of Hematoidin.—These occur as small yellow needles, rhombic plates or tufts, and are found in sputa which contain altered blood. They may be observed in abscess, gangrene, and cancer of the lungs.

Tubercle Bacilli.—The presence of tubercle bacilli in the sputum is an absolute proof of tuberculosis, but a failure to detect them after one or two examinations is no proof against phthisis. The bacillus is a fine rod, in length about half the diameter of a red blood-corpuscle, and often slightly bent and beaded. Its detection depends on its power, when stained, of resisting the bleaching effect of acids. To view it successfully, a $\frac{1}{12}$ inch oil-immersion lens is required.

Gabbett's Method.—Select with a clean needle one of the minute caseous masses contained in tuberculous sputum, spread it out in a very thin film on a cover-glass, dry in the air, and coagulate the albumin in the bacteria by passing the cover-glass, smeared side up, three times through the flame. Cover the specimen with Ziehl's carbol-fuchsin solution (fuchsin 1; alcohol 10; 5 per cent. aqueous solution of carbolic-acid crystals 90), and hold the cover-glass over the flame for a few minutes at such a distance that steam is formed. Wash off the excess of stain in water, and counterstain by treating the preparation for thirty seconds with Gabbett's solution (methylene-blue 2; sulphuric acid 25; water 75). Again wash in water, dry, and mount in Canada balsam. The tubercle bacilli will appear as red rods in a blue field.

PHYSICAL EXAMINATION OF THE RESPIRATORY ORGANS.

Inspection.—Inspection determines the shape of the chest, any unnatural prominence or depression, the amount of expansion, and any inequality of expansion.

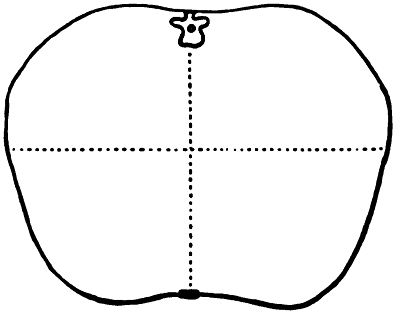

FIG. 10.—An outline of the normal chest.

Phthisinoid Chest.—The anteroposterior diameter is short; the thorax is long and flat; the ribs are oblique; the scapulæ are prominent; the spaces above and below the clavicles are depressed; and the angle formed by the divergence of the costal margins from the sternum is very acute.

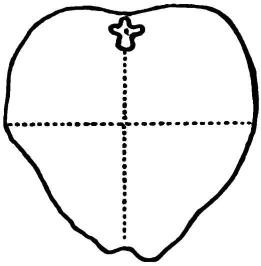

FIG. 11.—Rachitic chest.

Rachitic Chest.—This may resemble the former, but usually the sides are considerably flattened and the sternum prominent, so that the term "pigeon-breast" has been applied to this particular form. The sternal ends of the ribs are enlarged or "beaded," and this characteristic has given rise to the term "rachitic rosary." There is often a circular constriction of the thorax at the level of the xiphoid cartilage (Harrison's groove).

Emphysematous Chest.—In advanced emphysema the thorax is short and round; the anteroposterior diameter is often as long as the transverse; the ribs are horizontal; the angle formed by the divergence of the costal margin from the sternum is very obtuse or quite obliterated. The term "barrel-shaped chest" is applied to this configuration.

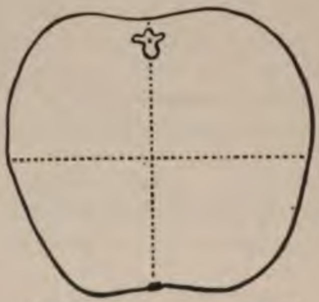

FIG. 12.—Emphysematous chest.

Local Prominences and Depressions.—An unnatural prominence or depression is often observed over the lower part of the sternum, and is generally congenital. The term "funnel breast" or "shoemaker's breast" (because it may result from the pressure of tools) has been applied to the sternal depression.

A unilateral or local depression may be due to: (1) Chronic phthisis; (2) cirrhosis of the lung; (3) pleurisy with fibrous adhesions.

A unilateral or local prominence may be due to: (1) Pleurisy with effusion; (2) pneumothorax, hydrothorax, hemothorax; (3) an aneurysm or tumor; (4) compensatory emphysema, resulting from impairment of the opposite lung;

(5) cardiac enlargements (left side); (6) enlargements of the abdominal organs, especially the liver and spleen.

Expansion.—In women and in children, and in both sexes during sleep, breathing is largely thoracic or costal; in men and in the old of both sexes it is largely abdominal or diaphragmatic.

Restricted abdominal breathing is observed in pregnancy, in abdominal tumors, and effusions; in peritonitis; in diaphragmatic pleurisy; in paralysis of the phrenic nerve from pressure or from bulbar disease; and occasionally in the "hysteric abdomen."

Diminished expansion of one side is observed in pleural effusions; in acute pleurisy (from pain); in consolidation of the lung from tuberculosis, pneumonia, or tumor; in occlusion of a bronchus; and in marked enlargement of the liver or spleen.

Increased expansion of one side is observed in compensatory emphysema.

Litten's Diaphragm Phenomenon.—When a healthy individual is placed in a horizontal position with the feet toward the window and all cross-lights are excluded, a narrow shadow may be seen descending between the sixth and the ninth rib in each axilla during full inspiration. It is due to the separation of the diaphragmatic pleura from the costal pleura during the inspiratory descent of the diaphragm. This shadow is absent in pleural effusion, pneumonia of the lower lobe, well-developed emphysema, and extensive pleuritic adhesions. As it is present in enlargement of the liver and in subphrenic abscess, it often aids materially in determining whether disease is above or below the diaphragm.

Palpation.—Palpation serves to detect any thoracic tenderness, edema, friction fremitus, or râles, and to determine the vocal fremitus and amount of expansion.

Thoracic tenderness is observed in pleurisy; in phthisis and pneumonia from being associated with pleurisy; in pleurodynia; in intercostal neuralgia (confined to certain spots); and in surgical affections, like caries and fracture of the ribs; and in contusion and inflammation of the parietes.

Edema of the chest-walls is recognized by "pitting" when pressure is made with the finger. It may be observed in empyema; in deep-seated abscesses of the parietes; after the application of a blister; and in general dropsy.

Friction Fremitus and Râles.—The friction-rub of pleurisy and harsh, sonorous râles can sometimes be detected by palpation.

Vocal fremitus is the sense of vibration imparted by the voice to the palpating hand.

In determining the vocal fremitus observe the following precautions: Palpate symmetric parts of the chest; make firm pressure; when comparing, use the same pressure on the two sides; apply the hands as nearly parallel to the ribs as possible; and remember that the fremitus is normally stronger over the right chest than the left.

The fremitus is usually slight in women and in children, and in men with thick chest-walls and a weak voice.

Vocal fremitus is abnormally increased when the lung is consolidated and the bronchi are patulous, as in—(1) Tuberculosis; (2) croupous pneumonia; (3) bronchopneumonia.

Vocal fremitus is decreased or absent in—(1) Pleural effusions—air, serum, pus, blood, or lymph; (2) emphysema; (3) pulmonary collapse from an obstructed bronchus; (4) pulmonary edema; (5) morbid growths of the lung.

Percussion.—Percussion determines resonance, pitch, and resistance.

Immediate percussion is performed by striking the chest directly with the fingers. It is not often employed, except over the clavicles, where the bones themselves act as pleximeters.

Mediate percussion is performed by using the fingers of one hand as a plexor and those of the opposite hand as a pleximeter; or by using a piece of ivory, glass, or hard rubber as a pleximeter, and a small hammer as a plexor.

The use of the fingers alone is preferable, for only in this way can resistance be determined.

In percussion the following precautions should be observed: Place the finger that is being used as a pleximeter firmly against the chest, and preferably parallel to the ribs; make the finger that is used as plexor strike the one on the chest perpendicularly; fix the forearm, and use no more force than can be obtained from a gentle swing of the wrist. When possible, percuss all parts of the chest anteriorly and posteriorly; percuss both in inspiration and in expiration. In comparing the two sides, be sure to percuss symmetric parts.

Normal Resonance.—On the right side, pulmonary resonance extends from a half inch to an inch above the clavicle, downward to the upper border of the sixth rib in front, and to a line drawn through the tenth spinous process posteriorly.

On the left side, pulmonary resonance extends from a half inch to an inch above the clavicle, downward, within the mammary line to the third rib, outside of the mammary line to the tenth rib, and posteriorly to a line drawn through the tenth spinous process.

Traube's Semilunar Space.—This is a tympanitic area at the base of the left chest, bounded above by the lung (sixth rib), on the right by the liver, and on the left by the spleen. It is obliterated in pleural effusion on the left side.

Hyperresonance is observed in the following conditions: (1) Pneumothorax. (2) Cavities—tuberculous or bronchiectatic. (3) Emphysema. (4) Lowered pulmonary tension, as above a pleural effusion or consolidation, and in the initial stage of pneumonia (Skoda's resonance). (5) Flatulent distention of the stomach or colon (frequently observed over the base of the left chest).

Tympanitic resonance is resonance of a hollow, drum-like character, like that normally obtained by percussing the empty stomach or the colon. It is elicited over the chest in pneumothorax and cavity-formation.

The **cracked-pot sound,** or *bruit de pôt fêlé*, is a modified tympany, and can be simulated by percussing over the cheek when the mouth is partially open. It may be normally heard over the chest of a crying infant (Walshe). In the adult it usually indicates a cavity that has a free communication with a bronchus. It is best detected by keeping the ear near the open mouth of the patient while percussing.

Dulness or flatness on percussion may be caused by the following conditions: (1) Pleural effusions of all kinds, except air; (2) consolidation of the lung from tuberculosis or pneumonia; (3) collapse of the lung; (4) congestion and edema of the lung; (5) morbid growths in the lung; (6) enlargement of the liver or spleen (at the bases).

Pitch.—Pitch of the note depends largely upon the volume of air, upon the tension of the walls of the cavity, and upon the size of the opening that communicates with the cavity. The less the air, the greater the tension, and the smaller the opening, the higher will be the pitch of the note. It is obvious, therefore, that conditions that are associated with hyperresonance may yield either a high- or a low-pitched note. In beginning phthisical consolidation the note over the affected apex is higher pitched; but it must be borne in mind that normally the note over the right apex is higher pitched than that over the left.

Resistance.—The sense of resistance appreciated by the percussing finger is increased in proportion as the air in the lung is decreased. It is generally more marked over a pleural effusion than over a consolidation with patulous bronchi.

Auscultation.—Auscultation of the lungs is practised to determine the character of the respiratory and voice sounds, and to detect adventitious sounds like râles.

In *immediate auscultation* the ear is placed directly over the chest, only a soft towel intervening.

In *mediate auscultation* the sounds are transmitted through a stethoscope, which should be applied to the bare chest.

In auscultation observe the following precautions: Do not exert much pressure with the stethoscope. When the chest is covered with hair, this should be moistened, otherwise it is likely to produce crackling sounds resembling râles. When possible, examine carefully all parts of the chest, anteriorly and posteriorly, during quiet breathing, during full inspiration, during full expiration, and after coughing. Compare carefully the sounds elicited over symmetric parts of the chest.

Normal Respiration.—Vesicular breathing is heard over the body of the lungs and is characterized by a soft, breezy inspiratory sound, and a shorter, lower pitched, less intense expiratory sound. Normally, the expiratory sound is not more than one-third the length of the inspiratory sound. Not infrequently expiration is wholly inaudible. Over the trachea and main bronchi the sounds are harsh and blowing, and the expiratory sound is as long as the inspiratory or slightly longer (bronchial breathing).

Modifications of the Respiratory Murmur.—*Puerile Breathing and Exaggerated Breathing.*—Normal breathing in children is called puerile breathing. Both inspiration and expiration are proportionately increased in length and loudness. Exaggerated breathing has the same characteristics as puerile breathing, and is heard after exertion and over the whole of one lung (compensatory emphysema) when the other is disabled.

Bronchial or Tubular Breathing.—This is harsh, blowing breathing with prolonged, accentuated expiration. The expiration may be considerably longer than the inspiration. and there is often a distinct pause between the two sounds. Bronchial breathing is heard normally over the trachea and in the interscapular space over the large bronchi, and abnormally over consolidated lung, when the bronchi are free. Thus, it may be heard in croupous pneumonia, bronchopneumonia, tuberculosis of the lung, and in pleurisy over the compressed lung.

Bronchovesicular Breathing.—This is breathing that is neither bronchial nor vesicular. It is somewhat harsh, and expiration is slightly prolonged and high pitched. Unlike puerile or exaggerated breathing, the normal ratio of inspiration to expiration is not maintained—the expiration is not relatively but *actually* prolonged. Bronchovesicular breathing may be heard when the lung is slightly solidified, as in beginning phthisis.

Cavernous or Amphoric Breathing.—This resembles bronchial breathing, but the sounds have a hollow character, and the pitch of the expiration is lower than that of inspiration. It may be imitated by blowing over the mouth of an empty jar.

Cavernous breathing may be heard in the following conditions: (1) Phthisical or bronchiectatic cavities; (2) pneumothorax, when the opening in the lung is patulous; (3) areas of consolidation near a large bronchus; (4) sometimes over lung compressed by a moderate effusion.

The Breathing of Emphysema.—This is weak breathing, with prolonged low-pitched or inaudible expiration.

Cogged-wheel or Jerky Breathing.—The respiratory murmur is not continuous, but is broken into waves. It is not indicative of any special disease, but it is frequently observed in hysteria, pleurodynia, bronchitis, and incipient phthisis.

Weak or Shallow Breathing.—This is noted: (1) When the chest-walls are thick; (2) in the old and feeble; (3) in emphysema; (4) in pleural effusion; (5) sometimes in incipient phthisis; (6) in painful affections of the chest, like pleurodynia and beginning pleurisy; (7) in pulmonary edema.

Vocal Resonance.—This is the confused humming sound heard over the chest when the patient speaks. It is modified by the same conditions that modify the vocal fremitus (see p. 208).

Bronchophony.—This is exaggerated vocal resonance. It is heard normally over the trachea, and abnormally over consolidated lung (phthisis and pneumonia) when the bronchi are free, over lung that is compressed by pleural effusion, and over some cavities.

Pectoriloquy.—This is a modification of vocal resonance in which the articulate speech is heard very distinctly, as though coming directly from the chest into the ear. It is more pronounced when the patient whispers.

Pectoriloquy is heard over: (1) Cavities that communicate with a bronchus; (2) areas of consolidation in the neighborhood of a large bronchus; (3) pneumothorax, when the opening in the lung is patulous; (4) some pleural effusions.

Egophony.—This is a modification of bronchophony, in which the sounds have a trembling or bleating quality. It is usually heard over slight pleural effusions near the upper border of dulness, especially near the inferior angle of the scapula.

Adventitious Sounds.—These are not modifications of preëxisting sounds, but wholly new sounds produced in the lung or pleura. They include râles, the friction-sound, metallic tinkling, and succussion-splash.

Râles.—These are new sounds created in the trachea, bronchi, air-vesicles, or in cavities. They may be due to the passage of air through liquid, through constricted tubes, or into collapsed air-vesicles.

- Pulmonary râles
  - Bronchial
    - Dry
      - Sibilant.
      - Sonorous.
    - Moist
      - Subcrepitant.
      - Bubbling.
      - Gurgling.
  - Vesicular = Crepitant.

Dry râles are probably produced by the passage of air over very viscid secretion in the tubes, although they have been ascribed to the passage of air through bronchial tubes that are narrowed by spasm or by swelling of the mucosa. They are heard particularly in bronchitis and asthma. Sibilant râles are whistling and high pitched; sonorous râles have a humming quality and are lower pitched. Dry râles may be heard on inspiration, expiration, or on both.

Moist râles result from the presence of liquid in the tubes; the thinner the liquid and the larger the tube, the coarser will be the râles. They may be heard on inspiration, expiration, or on both.

Subcrepitant or *crackling râles* are fine moist râles. They are heard in all conditions that are associated with liquid in the smaller tubes, as bronchitis, capillary bronchitis, pulmonary edema, and beginning phthisis.

Bubbling râles are coarser than subcrepitant, and are heard in bronchitis, in resolving croupous pneumonia, over phthisical deposits that are softening, and over small cavities.

Gurgling râles are very coarse, and resemble the bursting of large bubbles. They are heard over large cavities that contain fluid, and over the trachea in the so-called "death-rattle."

Crepitant Râles.—These are very fine râles, usually heard at the end of full inspiration. They may be simulated by rubbing a lock of hair between the fingers. They have been especially associated with the first stage of croupous pneumonia, and it has been supposed that they were due to the forcible separation of adherent vesicular walls. Râles very similar to, if not identical with, these are heard in pulmonary edema.

Friction-sounds are produced by the rubbing together of roughened pleural surfaces. They may be heard both in inspiration and in expiration, and often resemble subcrepitant râles, but they are more superficial and localized than the latter, and are not modified by cough or deep inspiration.

A roughened pleura in the neighborhood of the heart may produce a friction-sound of cardiac rhythm, and one which will still continue when the breath is held; under other conditions pleural friction-sounds cease when respiration is suspended.

Metallic Tinkling.—This name is applied to silvery or bell-like sounds that are heard at intervals over a pneumohydrothorax or large cavity. Speaking, coughing, and deep breathing usually induce them. Care must be taken not to confound them with similar sounds produced by the presence of liquid in a distended stomach.

Succussion-splash or *Hippocratic Succussion.*—This is a splashing sound produced by the presence of air and liquid in the chest. It may be elicited by gently shaking the patient while auscultating. It is almost pathognomonic of hydropneumothorax or a pyopneumothorax.

A similar splashing sound is often heard over a dilated stomach.

Mensuration.—In measuring the sides of the chest observe the following precautions: Measure from the middle of the sternum to the spinous processes; measure both sides after inspiration and after expiration; apply the tape with equal firmness to the two sides. In comparing, measure corresponding levels, and remember that the right side is from half an inch to an inch greater in circumference than the left.

The conditions that render one side more prominent than the other have already been considered.

Radioscopy.—In certain conditions of the chest radioscopy, especially the fluoroscopic screen, furnishes valuable information. It has been found of most service in detecting *aneurysms* and in determining the outlines of the heart in *emphysema.* Some observers have found it useful in detecting *tuberculous consolidation* before any other signs could be elicited. In *pleural effusion* the affected side is dark, the displacement of the heart is well shown, and the diaphragm appears flattened and depressed.

DISEASES OF THE NOSE AND LARYNX.

CORYZA.

(**Acute Rhinitis; Cold in the Head.**)

Definition.—An acute inflammation of the nasal cavities.

Etiology.—Exposure to cold and to wet, especially when the body is overheated, is a common cause. It may be excited by the inhalation of irritating vapors or dust. It is an expression of iodism. It is a symptom of certain infectious diseases—especially of measles and influenza.

Pathology.—The mucous membrane is red and swollen. In the first stage there is no secretion, but later irritating, watery mucus flows from the nose and excoriates the lip; this in time is followed by a copious mucopurulent discharge.

Symptoms.—The disease is ushered in with chilliness, malaise, fulness in the head, and sneezing. The nasal chambers are obstructed, so that the patient is obliged to breathe through his mouth. At first there is no secretion, but in twenty-four or forty-eight hours a watery discharge is established, which later becomes mucopurulent. Slight fever and its associated symptoms are commonly present. The duration is from a few days to two weeks.

Complications.—The disease is not infrequently accompanied with conjunctivitis, pharyngitis, laryngitis, and catarrh of the Eustachian tube and middle ear that results in temporary deafness.

Prognosis.—Favorable.

Treatment.—When the patient is seen at the outset and is willing to remain indoors for twenty-four hours, a hot foot-bath, with a full dose of Dover's powder, followed in the morning by a Seidlitz powder or other saline aperient, often gives excellent results. When the patient is fully able to go about, the following capsules will usually afford considerable relief:

℞. Pulveris camphoræ . . . . gr. vj
Extracti belladonnæ
Codeinæ sulphatis . . . . āā gr. iss
Cinchoninæ sulphatis . . . . gr. xij.—M.
Pone in capsulas No. xij.
SIG.—One every two or three hours.

Warm Dobell's solution (see p. 37) or warm distilled extract of witch-hazel (diluted with 1 part of water) used as a spray at intervals, and followed in a few minutes by an oily application like the following, generally renders satisfactory service:

℞. Mentholis . . . . gr. iij
Olei pini pumilionis . . . . ♏ v
Petrolati liquidi . . . . . . q. s. ad f℥j.—M.

CHRONIC NASAL CATARRH.

(Chronic Rhinitis.)

Definition.—A chronic inflammation of the nasal mucous membrane.

Etiology.—Repeated attacks of acute coryza, impure air, the continual inhalation of irritating dusts or vapors, lowered vitality, and congenital or acquired obstruction of the nasal chambers are causal factors. It is sometimes an expression of syphilis.

Varieties.—(1) Simple chronic rhinitis; (2) hypertrophic rhinitis; (3) atrophic rhinitis.

Symptoms.—These consist in a mucoid or mucopurulent discharge from the nose; obstruction of the nostrils from swelling or hypertrophy of the mucosa or from inspissated secretion; mouth-breathing; a nasal intonation of the voice; frontal headache; and impairment of the sense of smell.

Symptoms of catarrh of the neighboring organs are frequently present. The most common of these are: dryness of the throat and hawking from pharyngitis; deafness from catarrh of the middle ear; and watering of the eyes from catarrhal occlusion of the lacrimal canal.

Simple Chronic Rhinitis.—The mucous membrane of the nose is congested, swollen, and highly irritable. There is hypersecretion of mucus or mucopus.

Hypertrophic Rhinitis.—The mucous membrane is red and the cavities are more or less occluded from hypertrophy of the cavernous tissue covering the turbinated bones. In advanced cases exostoses from the bony framework are sometimes noted. The secretion is usually composed of thick mucopus. Adenoid growths are often found in the nasopharynx.

Atrophic Rhinitis (Ozena).—This form is seen most frequently in young adults, and is more common in women than in men. The nasal chambers are large; the mucous membrane is pale, dry, and glazed; adherent scabs are generally present. The secretion is very abundant, thick, and of a yellowish or greenish color. A characteristic feature is the extremely offensive odor, which is probably due to the decomposition of the retained secretion. In advanced cases there may be necrosis of the bones and sinking in of the bridge of the nose.

Prognosis.—In the simple and hypertrophic form the prognosis is favorable under persistent treatment. In atrophic rhinitis perfect cure is rarely attainable, but great improvement is possible.

Treatment.—Any constitutional vice, if present, should receive appropriate treatment. Fresh air, outdoor exercise, and frequent bathing, with friction of the skin, are to be recommended. Tonics, especially strychnin and cod-liver oil, are often required. The nasopharynx must be kept clean by means of antiseptic sprays, such as Dobell's solution (see p. 37) or the following:

℞.	Sodii bicarbonatis		
	Sodii boratis	āā	gr. xxx
	Sodii chloridi		gr. iij
	Thymolis		
	Mentholis	āā	gr. ss
	Olei gaultheriæ		♏ ij
	Glycerini		f℥ij
	Alcoholis		f℥ss
	Aquæ	q. s. ad	f℥viij.—M.

In the hypertrophic form local remedies of an astringent or alterative character are often efficacious. The following are in common use: A mixture of iodin and glycerin containing 6 grains of iodin, 12 grains of potassium iodid, and 1 ounce each of glycerin and water; aqueous solution of ichthyol (20 to 40 per cent.); solution of zinc sulphocarbolate (2 to 5 per cent.); and solution of silver nitrate (1 to 2 per cent.).

When the hypertrophic process proves resistant, the obstruction must be removed by means of caustics (chromic or trichloracetic acid), the galvanocautery, or the snare.

In atrophic rhinitis the crusts may be removed by pledgets of cotton soaked in a solution of hydrogen dioxid. After the nares have been thoroughly cleansed, an oily solution like the following may be applied:

℞.	Mentholis	gr. xx
	Thymolis	gr. vj
	Eucalyptolis	♏xx
	Petrolati liquidi	f℥vj.—M.

For destroying the offensive odor one of the following applications may be used: Pledgets of cotton soaked in an aqueous solution of ichthyol (20 to 50 per cent.); sprays of formalin (1 : 1000), of Labarraque's solution (1 : 30), or of potassium permanganate (2 grains to the ounce).

ACUTE CATARRHAL LARYNGITIS.

Definition.—An acute catarrhal inflammation of the larynx.

Etiology.—Improper use of the voice, exposure to cold and wet, and the inhalation of irritating dusts or vapors are its common causes. It may be excited by the impaction of a foreign body. It is also an associated condition in certain infectious diseases, like whooping-cough, measles, diphtheria, and influenza.

Symptoms.—The chief symptoms are: Hoarseness of the voice or aphonia; hard, ringing cough; pain in the throat, increased by speaking, coughing, and swallowing; expectoration, which is at first scanty and later mucopurulent;

fever and its associated symptoms. In sensitive persons, and especially in children, paroxysms of croupy cough and dyspnea (*false croup*) may result from spasm of the vocal cords. When there is much edema, severe dyspnea becomes a prominent feature.

Inspection.—The mucous membrane of the laryngeal walls and vocal cords is red and swollen. In grave cases the tissues are highly edematous.

Prognosis.—In simple laryngitis without edema the prognosis is altogether favorable. The attack usually lasts from a week to ten days. When there is edema of the larynx, indicated by dyspnea or threatened asphyxia, the prognosis is grave.

Treatment.—Use of the voice should be avoided. The air of the room should be rendered moist by means of steam. An ice-bag or iodin may be applied externally. At the onset it is advisable to administer a mild aperient. Mild expectorants—ipecac, potassium citrate, or ammonium chlorid—may be given in conjunction with heroin, codein, or paregoric when the cough is troublesome. Such a combination as the following is often serviceable:

℞.	Potassii citratis	ʒij
	Vini ipecacuanhæ	fʒiv
	Tincturæ opii camphoratæ	fʒiv
	Syrupi tolutani	f℥iss
	Aquæ	q. s. f℥iv.—M.

SIG.—A dessertspoonful every three hours.

In acute *edematous laryngitis*, when the swelling does not yield promptly to local bloodletting, the external application of ice, astringent sprays, scarification of the mucous membrane, and active catharsis, tracheotomy should be performed without delay.

SPASMODIC CROUP.

(False Croup; Catarrhal Croup.)

Definition.—Spasm of the vocal cords, excited by catarrh of the larynx.

Etiology.—The attacks usually occur in young children, and are induced by the causes of catarrhal laryngitis.

Symptoms.—Generally there has been a little hoarseness and cough during the day, and at night the child is awakened from sleep by a severe paroxysm of suffocative cough. The latter has a peculiar, hard, metallic quality, and is associated with the evidences of dyspnea, namely: anxious face, dilating nostrils, prominent sternocleidomastoids, and retraction of the base of the chest with each inspiratory effort. During the paroxysm the skin is hot and the pulse is tense and rapid. In from a few moments to an hour the cough ceases, free perspiration follows, and the child falls asleep.

Two or three similar attacks may occur in the same night, but on the following day the child appears quite well. A recurrence of the seizures for several successive nights is not infrequent.

Diagnosis.—**Membranous or Pseudomembranous Croup (Diphtheria).**—Hoarseness and dyspnea develop gradually, and the latter is not intermittent. False membrane may be seen in the throat or may be coughed up. The constitutional symptoms are more severe.

Laryngismus Stridulus.—This is a pure neurosis, and is often associated with the rachitic diathesis. The paroxysms resemble those of false croup, but are associated with a peculiar crowing inspiration, and lack catarrhal symptoms, such as hoarseness and cough.

Prognosis. —Always favorable.

Treatment.—A sponge moistened with hot water may be applied to the throat, or the child may be placed in a hot bath. If these simple measures fail, an emetic will almost invariably bring relief. Wine of ipecac (1 dram) may be selected. Subsequent treatment should be directed to the laryngeal catarrh. A mixture like the following will be found useful:

℞.	Tincturæ aconiti	♏xx
	Vini ipecacuanhæ	fʒj
	Potassii bromidi	ʒss
	Potassii citratis	ʒj
	Syrupi tolutani	f℥j
	Aquæ	q. s. ad f℥ij.—M.

SIG.—A teaspoonful every two or three hours for a child of two years.

MEMBRANOUS CROUP.

(Croupous Laryngitis; True Croup; Pseudomembranous Laryngitis.)

See Laryngeal Diphtheria.

CHRONIC LARYNGITIS.

Simple Chronic Catarrhal Laryngitis.—This may follow an acute attack, or it may develop gradually from overuse of the voice, excessive smoking, or inhalation of dust or irritant vapors.

Symptoms.—These consist in moderate hoarseness, aphonia after continued speaking, slight cough, and scanty expectoration of grayish mucus tinged with dust or other impurities.

Laryngoscopic examination reveals redness and swelling of the vocal cords or of the entire larynx.

Treatment.—The use of the voice should be restricted. Coexisting nasal or pharyngeal disease should receive attention. The patient must learn to use the voice properly, expelling sounds by the abdominal muscles and diaphragm, and not by the muscles of the throat. Flannel protectors should be avoided, and the application of cool water to the neck, night and morning, instituted in their stead. Tonics are frequently indicated.

Thorough cleansing of the nose, throat, and larynx should be secured by means of mild alkaline sprays (Dobell's solution). Astringent sprays like the following are useful: Zinc acetate, 3 to 5 grains to the ounce; zinc sulphocarbolate, 2 to 3 grains to the ounce; alum, 3 to 5 grains to the ounce. Direct applications of silver nitrate (3 to 5 grains to the ounce) are also very efficacious.

Tuberculous Laryngitis.—This may be primary, but it is more often secondary to tuberculosis elsewhere, especially the lung.

Symptoms.—These consist in hoarseness, aphonia, hacking cough, and pain in the throat, increased by coughing, speaking, and swallowing.

Laryngoscopic Examination.—The mucous membrane is swollen, *pale*, and edematous. The arytenoid cartilages are especially involved, and the membrane between them is often the seat of a hill-like infiltration. Tuberculous ulcers are usually shallow and have a broad base, an irregular outline, and an uneven surface. They are extremely painful.

Treatment.—The general treatment should be that of pulmonary tuberculosis. The parts should be frequently cleansed with alkaline detergent sprays. Terebene, compound tincture of benzoin, or eucalyptol may be used in a respirator or inhaled from the surface of boiling water. Radical treatment consists in rubbing in, under cocain anesthesia, lactic acid (30 to 75 per cent. solutions). Palliative treatment consists in applying cocain in solution (4 to 10 per cent.) or orthoform, morphin, or iodoform in powder. The following insufflation is useful:

℞.	Orthoform	gr. j
	Acidi borici	gr. j
	Morphinæ sulphatis	gr. ⅛.

Syphilitic laryngitis may manifest itself as a catarrhal inflammation, mucous patches, gummatous infiltration, or ulceration. The ulcers are more or less circular, deep, and sharply circumscribed. They are frequently found on the epiglottis. Rapid necrosis and exfoliation of the cartilage may follow. Pain is often slight.

Diagnosis.—The history, the presence of other syphilitic lesions, the deep, clean-cut, rapidly spreading ulcers, the effect of treatment, and the absence of marked pain and of pulmonary lesions well serve to distinguish syphilis from *tuberculosis.*

Treatment.—Constitutional treatment with iodids and mercurials is of the first importance. Local cleanliness should be secured by thorough spraying with some alkaline antiseptic solution. Ulcers may be touched with silver nitrate (melted on a silver probe), acid nitrate of mercury (1 to 5 parts of water), or chromic acid (1 to 8 parts of water). Insufflations of iodoform are also useful. Cicatricial stenosis may call for gradual dilatation or even tracheotomy.

LARYNGISMUS STRIDULUS.

(Spasm of the Glottis; Laryngospasmus; "Child-crowing.")

Definition.—A paroxysmal neurosis, characterized by reflex spasm of the adductors of the larynx, and not excited by any local inflammation.

Etiology.—It usually occurs in rachitic infants between six months and two years of age. The attacks may be brought on by fright, gastric irritation, exposure, or a sudden movement.

Symptoms.—The attacks often occur on waking from sleep, and are characterized by a sudden arrest of breathing and tonic muscular spasms. The face is pale, and later cyanosed; the eyes are rolled up; the body is arched; the thumbs are turned into the palms; the legs are extended, and the soles turned inward. In a few seconds the spasm relaxes, and air is drawn through the glottis with a shrill, crowing sound.

The seizures vary greatly in frequency: several may occur in a day, or they may be weeks apart.

Diagnosis.—The intermittent character of the affection; the peculiar crowing inspiration; the absence of fever, cough, and hoarseness will serve to distinguish laryngismus from *croup*.

Prognosis.—Favorable. In the very young death may result from suffocation.

Treatment.—*The Paroxysm.*—Cold water may be dashed on the face and head, or a few drops of nitrite of amyl may be placed on a handkerchief and held before the nose.

The Interval.—Careful search should be made for some exciting cause; the gums may require lancing, or the gastro-intestinal tract may demand attention. The child should be placed under the best hygienic conditions. The food should be plain and nutritious; tonics, like cod-liver oil, hypophosphites, and arsenic, are generally indicated. The bromid of sodium is an efficient antispasmodic, and may be advantageously combined with antipyrin:

℞.	Sodii bromidi	ʒiss
	Antipyrinæ	gr. xx–xxx
	Glycerini	f℥ss
	Aquæ menthæ piperitæ	f℥iij.

SIG.—A teaspoonful four times a day.

EDEMA OF THE LARYNX.

(Edema of the Glottis.)

Definition.—An infiltration of serous fluid into the submucous tissue of the larynx.

Etiology.—It occasionally results from severe attacks of catarrhal laryngitis. It may be induced by severe inflammation of neighboring organs—as the tonsils, parotid glands, and pharynx. It may be a complication of some acute infectious disease—like diphtheria, scarlet fever, or facial erysipelas. It is sometimes associated with ulcerative affections of the larynx, like tuberculosis and syphilis. It may be excited by the irritation of burns, scalds, or caustics. It occasionally occurs abruptly in the course of Bright's disease.

Pathology.—The connective tissue of the larynx is infiltrated with a serous or seropurulent fluid. The mucous membrane is tense and changed in color.

Symptoms.—These are: hoarseness of the voice, and later aphonia; extreme dyspnea, at first on inspiration, but later on expiration also; stridulous respiration; barking cough; and the evidences of dyspnea—namely, anxious face, protruding eyes, blue lips, prominent sternocleidomastoids, and retraction of the base of the chest. When the epiglottis is involved, the swelling can be detected by the finger in the throat.

Laryngoscopic Examination.—The mucous membrane is swollen and of a reddish-purple color. The epiglottis may resemble a round, translucent tumor. In infraglottic edema the upper part of the larynx may appear normal, but swollen and edematous membrane is seen projecting through the glottis. The vocal cords are rarely affected.

Prognosis.—Extremely grave.

Treatment.—Mild inflammatory edema sometimes yields to the sucking of ice, local bloodletting, the application of ice to the neck, astringent sprays (alum, adrenalin, tannic acid), and the administration of saline purges. When the symptoms become urgent, the parts should be scarified under cocain anesthesia, and if this fails, tracheotomy should be performed at once.

DISEASES OF THE LUNGS.

BRONCHITIS.

Definition.—An inflammation of the bronchial tubes, characterized by substernal soreness, cough, mucopurulent expectoration, and dry and moist râles.

Varieties.—(1) Acute catarrhal bronchitis; (2) chronic catarrhal bronchitis; (3) fibrinous bronchitis.

ACUTE CATARRHAL BRONCHITIS.

Etiology.—A cold, damp climate; changeable weather; occupations that necessitate confinement or the inhalation of irritating dusts or vapors; the gouty diathesis; and chronic heart disease are general predisposing factors.

Exposure to cold and wet, particularly when the body is overheated, or the inhalation of irritating gases or dusts is the usual exciting cause. Acute bronchitis is also an associated condition in certain infectious diseases, especially measles, whooping-cough, typhoid fever, and influenza.

Micro-organisms (streptococcus, staphylococcus, and pneumococcus) are without doubt important etiologic factors.

Pathology.—In most cases the trachea and large tubes only are affected. The mucous membrane is red, swollen, injected, and more or less covered with tenacious mucopus.

Microscopic examination reveals desquamation of epithelium and infiltration of the submucous tissues with leukocytes.

Symptoms.—The chief features are: Chilliness; malaise; a sense of soreness and constriction behind the sternum, which is increased by coughing; slight fever (100° to 102° F.), with its associated symptoms; and cough, which is at first dry and painful, but later accompanied by free mucopurulent expectoration.

Physical Signs.—Inspection, palpation, and percussion usually give negative results.

Auscultation at first reveals sibilant and sonorous râles on both sides of the chest, and in the second stage, when secretion is established, moist râles.

Diagnosis.—**Influenza.**—High fever, severe pain in the head, back, and limbs, and great prostration will serve to distinguish influenza from bronchitis when the former is prevalent.

Catarrhal Pneumonia.—Moderately high and irregular fever, prostration, pronounced dyspnea, cyanosis, and physical signs indicating consolidation will aid in the recognition of pneumonia.

Prognosis.—Favorable. In the old, young, and feeble there is danger of its leading to capillary bronchitis or catarrhal pneumonia. The duration is from one to three weeks.

Treatment.—If the patient be weak or old, he should be confined to his room or even to bed; the atmosphere of the room should be kept warm and moist. If the patient be seen at the outset, it is useful to promote free diaphoresis, and this may be accomplished by means of a hot foot-bath, with hot drinks and a full dose of Dover's powder. Counterirritation to the chest in the form of sinapisms or stupes is very beneficial. The food should be simple and readily digestible, and the bowels should be kept regularly open by the aid of mild aperients. In the early stage, when there is no secretion, sedative expectorants—ipecac, potassium citrate, tartar emetic, and apomorphin—are indicated. It is usually necessary to add a sedative, like opium or one of its derivatives (codein, $\frac{1}{8}$ to $\frac{1}{6}$ grain, or heroin, $\frac{1}{16}$ to $\frac{1}{10}$ grain) to allay the distressing cough. A combination like the following will be found useful:

℞. Potassii citratis . . . . ʒiij
Vini ipecacuanhæ . . . . fʒiiss
Tincturæ opii camphoratæ . . . . . fʒiij
Succi limonis . . . . f℥j
Syrupi . . . . q. s. f℥vj.—M.
SIG.—A tablespoonful every four hours.

When the secretion becomes more abundant, stimulant expectorants are indicated. One of the most reliable members of this class is ammonium chlorid; it may be prescribed in some simple vehicle, like brown mixture, or with squills, as in the following formula:

℞. Ammonii chloridi . . . . ʒiss
Syrupi scillæ . . . . fʒv
Tincturæ opii deodorati . . . . ♏xl
Extracti glycyrrhizæ . . . . ʒj
Glycerini . . . . f℥ss
Aquæ . . . . q. s. ad f℥iv.—M.
SIG.—A dessertspoonful in water every four hours.

Among other useful stimulant expectorants may be mentioned terpin hydrate, terebene, oil of eucalyptus, oil of santal, and tar. Such combinations as the following are often serviceable when the catarrh tends to become subacute:

℞. Terebini
Olei santali . . . . aa fʒss
Strychninæ sulphatis . . . . gr. ¼
Codein. sulphatis . . . . gr. ij–iij.—M.
Pone in capsulas No. xij.
SIG.—One every four hours.

Or:

℞. Ammonii chloridi . . . . ʒj
Vini picis liquidæ
Misturæ glycyrrhizæ compositæ . aa f℥iss.—M.
SIG.—A dessertspoonful every two or three hours. (MUSSER.)

In the aged and infirm alcoholic stimulants are often required to combat general adynamia. Strychnin is a most valuable adjunct to the expectorants when there are indications that the heart is becoming strained by the violent paroxysms of cough. Should there be evidence of pronounced cardiac failure, it will be necessary to employ digitalis.

Such tonics as cod-liver oil, iodid of iron, quinin, and arsenic are often useful during convalescence from severe and prolonged attacks. Much benefit will also be obtained from suitable change of climate.

CHRONIC BRONCHITIS.

(Chronic Bronchial Catarrh; Winter Cough.)

Etiology.—It may result from the continuation of an acute attack; but more frequently it develops gradually in association with gout or chronic heart or kidney disease. It is especially common in the old. It is an associated condition in emphysema, phthisis, chronic interstitial pneumonia, and in many cases of asthma.

Pathology.—The mucous membrane of the bronchi is sometimes thickened and roughened from an overgrowth of the connective tissue; in other cases the mucosa is thin from atrophic changes. The surface is usually covered with mucopus; ulcers are occasionally noted.

Long-standing bronchitis leads to dilatation of the tubes (bronchiectasis) and to emphysema.

Symptoms.—The chief features are: Persistent cough with more or less mucopurulent expectoration; a sense of soreness behind the sternum. Fever is usually absent, and unless the disease is very severe, the general health may be fairly well preserved. Dyspnea on exertion is sometimes a troublesome symptom; it, however, belongs more to the resulting emphysema than to the bronchitis.

Physical Signs.—Unless emphysema has developed, inspection, palpation, and percussion give negative results. Auscultation sometimes reveals râles, some of which are dry and wheezing, while others are moist and bubbling.

Bronchorrhea.—This term is applied to cases of chronic bronchitis which are associated with a very copious expectoration. The sputum is generally mucopurulent, and sometimes very offensive (fetid bronchitis.)

Dry Catarrh.—This form, described by Laennec as *catarrhe sec*, is characterized by severe spells of coughing that are accompanied by little or no expectoration. It is generally seen in the old in association with emphysema or asthma.

Diagnosis.—**Phthisis.**—The absence of fever, of hemorrhage, of bacilli in the sputa, and of signs indicating consolidation will serve to distinguish chronic bronchitis from phthisis.

Bronchiectasis.—This often results from chronic bronchitis. It is characterized by paroxysms of cough attended with the expectoration of large quantities of purulent secretion of an extremely offensive odor. There may be, also, physical signs of one or more cavities near the root or base of the lung.

Emphysema.—Marked dyspnea, distention of the chest, hyperresonance on percussion, and a prolonged feeble expiration on auscultation will indicate emphysema.

Sequelæ.—Emphysema, bronchiectasis, and dilatation of the right ventricle.

Prognosis.—Perfect recovery is rarely attainable, but the disease is not incompatible with long life.

Treatment.—Treatment must be directed toward the prevention of recurrent attacks, and the removal, if possible, of the underlying cause. Change of climate, especially in winter, is most beneficial. When there is much secretion, a dry, warm climate is generally to be recommended, whereas if there be little expectoration, a moist warm climate is preferable. When patients cannot afford to travel, they should remain indoors as much as possible in bad weather, and take every precaution against exposure. Flannel should at all times be worn next to the skin, the feet should be kept dry, and night air should be avoided.

Underlying chronic diseases should receive appropriate treatment. When cardiac insufficiency is present, digitalis or strychnin will be required. When there is general malnutrition, such remedies as iron, arsenic, cod-liver oil, and hypophosphites may be given with advantage. When gout is a factor, iodids and alkalis will prove serviceable.

The most useful *direct remedies* are the stimulant expectorants, such as terebene, oil of eucalyptus, myrtol, oil of santal, oil of copaiba, oil of cubeb, and tar. When the sputum is heavy and purulent, no drug acts so well as creasote or the carbonate of guaiacol. Potassium iodid may be tried when the expectoration is scanty and viscid. Mild anodynes—heroin or codein—are often necessary to control harassing cough. The following formulas will illustrate the manner in which these remedies may be combined:

℞. Terebini
Olei eucalypti
Olei santali . . . . aa fʒj–iss
Codeinæ . . . . gr. iij–vj.—M.
Pone in capsulas No. xxiv.
SIG.—One after each meal and at bedtime.

℞. Terpini hydratis . . . . ʒj
Guaiacolis carbonatis . . . . ʒij
Strychninæ sulphatis . . . . gr. ss
Codeinæ . . . . gr. iij.—M.
Pone in capsulas No. xxiv.
SIG.—One or two capsules three or four times a day.

℞. Apomorphinæ hydrochloridi . . . . . gr. ss
Syrupi pruni virginianæ . . . . f℥ij
Syrupi picis liquidæ . . . . f℥iv.—M.
SIG.—A tablespoonful thrice daily. (MURRELL.)

Inhalations (eucalyptol, terebene, oil of Scotch fir, compound tincture of benzoin, etc.) are often efficacious. Such a mixture as the following may be employed several times a day in an oronasal respirator:

℞. Chloroformi . . . . f℥ss
Creasoti
Terebini
Olei pini sylvestris . . . . . . aa fʒiss
Alcoholis . . . . q. s. ad f℥j.
SIG.—From 5 to 20 drops to be used in the inhaler several times a day.

Counterirritation, preferably with iodin or small blisters, is often of great service in lessening the severity of acute exacerbations.

FIBRINOUS BRONCHITIS.

(Croupous Bronchitis; Pseudomembranous Bronchitis.)

Definition.—A rare affection characterized by the expectoration of fibrinous casts of certain portions of the bronchial tree.

Ætiology.—The causes are unknown. In some cases it has been associated with tuberculosis, while in others there has been chronic heart disease.

Pathology.—The disease is often limited to a certain number of bronchi. Some of the affected tubes are found filled with a fibrinous exudate, while others are found empty and show a loss of epithelium. The casts are usually expelled in the form of whitish balls, which, when unrolled in water, present branching molds of the divisions and subdivisions of the affected bronchi. On close examination they are found to be hollow and laminated. Under the microscope, a homogeneous or fibrillated membrane is observed, imbedded in which are leukocytes, fat-drops, particles of pigment, epithelial cells, and occasionally Leyden's octahedral crystals.

Symptoms.—Acute and chronic forms are recognized. The former is rare, and manifests the symptoms of a severe attack of acute bronchitis, but the sputa contain fibrinous casts and there is marked dyspnea.

The chronic form is characterized by severe cough, paroxysms of dyspnea, and the expectoration of fibrinous plugs. Hemoptysis is not uncommon. The physical signs are those of chronic bronchitis and emphysema. The disease often lasts a few weeks, and then disappears, to return again at definite periods.

Prognosis.—In the acute variety the prognosis must be guarded; death frequently results from suffocation.

The chronic variety runs a very protracted course.

Treatment.—In the acute cases the atmosphere of the room should be kept moist and uniformly warm. Inhalations of alkaline vapors (lime-water) appear to be beneficial. Counterirritants should be applied to the chest. Emetics sometimes aid in the expulsion of loose casts. Sedative or stimulant expectorants may be prescribed, as in catarrhal bronchitis. In the chronic form potassium iodid may also be given.

BRONCHIECTASIS.

(Dilatation of the Bronchi.)

Definition.—A uniform or circumscribed dilatation of the bronchi.

Etiology.—It is most frequently the result of chronic bronchitis, weakening of the walls of the bronchi from the inflammation and increased pressure from the violent coughing being the determining factors. It is occasionally excited by obstruction to a bronchus from a foreign body or the pressure of an aneurysm. Finally, the contraction of overgrown connective tissue in fibroid pneumonia, chronic phthisis, and chronic pleural thickening sometimes induces marked ectasy.

Pathology.—Two forms are noted: (1) The cylindric form, in which the tubes, particularly those of medium size, are uniformly dilated in one or both lungs; and (2) the saccular form, in which the tubes swell out, here and there, into circumscribed dilatations that may reach several inches in diameter. Bronchiectatic cavities are lined with mucous membrane, but the latter is often atrophied, indurated, or ulcerated.

Symptoms.—The chief symptoms are paroxysmal cough, dyspnea, and copious expectoration. The last is characteristic; it is apt to occur periodically in gushes; the material has a highly offensive odor, and when allowed to stand in a glass vessel, separates into three layers: an upper layer of dirty brown froth, a middle layer of turbid mucus, and an under layer of decomposed pus. Microscopically, it contains pus-corpuscles, fat crystals, crystals of hematoidin, and numerous micro-organisms, but no tubercle bacilli. Elastic fibers are rarely found. Hemoptysis is not uncommon.

Physical Signs.—In the cylindric variety the signs are those of chronic bronchitis. The saccular variety may present the signs of tuberculous cavities—localized tympany, cavernous breathing, gurgling râles, and pectoriloquy. Bronchiectatic cavities are usually near the root or the base of the lung.

Diagnosis.—**Phthisis.**—The marked constitutional symptoms, the apical location of the cavities, the signs of consolidation around the cavities, and the presence of tubercle bacilli in the sputum will establish the diagnosis.

Prognosis.—There is little prospect of cure, but life may be prolonged indefinitely.

Treatment.—The general treatment is that of chronic bronchitis. The most useful expectorants are oil of eucalyptus, oil of santal, terebene, tar, guaiacol carbonate, and creasote. Inhalations of terebene, carbolic acid, creasote, etc., lessen cough and aid in destroying the fetid odor of the breath. When a single large cavity can be definitely located in the lower lobe, incision and drainage may be considered.

ASTHMA.

Definition.—Paroxysmal dyspnea due to spasm of the bronchi or to sudden swelling of the bronchial mucosa.

Etiology.—Asthma is a symptom of several diseases, but a hypersensitive condition of the mucous membrane of the respiratory tract appears to be essential to its production. When this condition prevails, asthma may be induced—(1) By the pulmonary congestion of cardiac disease (cardiac asthma); (2) by the uremic intoxication or transient pulmonary edema of Bright's disease (renal asthma); or (3) by some irritant from without, as the pollen of plants (hay-asthma); (4) sometimes the paroxysms are excited by the most trivial causes, as an atmospheric change or a peculiar odor, and to this form many writers restrict the term asthma. This last will be discussed under the head of Essential Asthma.

ESSENTIAL ASTHMA.

(Bronchial Asthma; Nervous Asthma; Spasmodic Asthma.)

Etiology.—Heredity, a neurotic temperament, and lesions of the upper air-passages (hypertrophic rhinitis, polyps, etc.) are predisposing factors. More males are affected than females. It may develop at any age. Atmospheric changes, the inhalation of certain kinds of dust, the odor of certain animals or plants, reflex irritation, indigestion, a change of locality, or bronchial catarrh may serve as an exciting cause.

Pathology.—The disease is a pure neurosis, and the paroxysms probably result from spasm of the bronchial muscles or a sudden vasomotor turgescence of the bronchial mucosa.

Symptoms.—The paroxysms often appear suddenly, but in some cases certain symptoms precede and give warning of the approaching attack; among these are chilliness, flatulence, sneezing, and a copious discharge of pale urine. The attacks most often occur at night. There is a sense of oppression and anxiety, followed by dyspnea so intense that the patient runs to the window for air, or sits upright with his arms in such a position that he can bring into play the auxiliary muscles of respiration. The face is pale and anxious, the lips are blue, and the surface is covered with profuse perspiration. The respirations are not rapid, but labored and noisy. Cough is usually present, and is associated with the expectoration of thick, tenacious mucus. On close examination little grayish plugs can be detected in the sputum. These, under a pocket-lens, are seen to consist of delicate spirals of mucus that have been molded in the finer bronchioles (Curschmann's spirals).

Microscopic examination also reveals eosinophile cells and octahedral crystals (Charcot-Leyden crystals).

The paroxysms may last from a few minutes to many hours, and may recur for several successive nights, or may disappear entirely for weeks or months.

Physical Signs.—*Inspection* reveals expiratory dyspnea and distention of the chest.

Percussion reveals hyperresonance.

Auscultation.—Vesicular breathing is weak and obscured by abundant sibilant and sonorous râles. The latter are especially marked in expiration, which is greatly prolonged and wheezy.

Diagnosis.—**Cardiac and renal asthma** are to be distinguished from essential asthma by the history and by the evidence of organic heart or kidney disease.

Hay-asthma may be recognized by the periodicity of the attacks and the associated coryza and sneezing.

Laryngeal Obstruction from Foreign Bodies, Croup, Paralysis of the Vocal Cords, or Edema.—The dyspnea is with inspiration, and the chest, instead of being distended, is retracted, especially at the base.

Sequelæ.—Emphysema invariably follows when the asthma is of long duration; it results from the tension to which the vesicles are subjected during the expiratory effort. Dilatation of the right ventricle is also a remote sequel.

Prognosis.—The disease does not prove fatal except through complications or sequelæ. Recovery is rare. Cases associated with some definite reflex irritation, as nasal obstruction, occasionally recover when the cause is removed.

Treatment.—*The Interval.*—The cause must be sought for in every case, and removed if possible. Digestive disturbances should always receive careful attention. Chronic bronchitis, emphysema, and dilatation of the heart are frequent concomitants of asthma and call for special treatment. Change of climate, even though slight, generally proves of decided service, but the choice of locality must be determined very largely by the personal experience of the patient. Many sufferers do better in the smoky atmosphere of cities than in the country, but a dry atmosphere with a moderate elevation is better suited for the majority.

Among internal remedies none has proved more useful than potassium iodid (5 to 10 grains thrice daily) in averting attacks. Tincture of belladonna (3 to 5 minims thrice daily) is a valuable adjunct to the iodid. Arsenic may be tried when the iodids fail. *Grindelia robusta* is sometimes useful when there is much catarrh. Strychnin is of service in cases associated with emphysema.

The Attack.—Some patients derive great benefit from the fumes of ignited stramonium or belladonna leaves or paper that has been impregnated with potassium nitrate. These agents may be burnt in the patient's room or smoked in a pipe or in the form of cigarets. Marked alleviation of the paroxysms is often obtained from the inhalation of amyl nitrite (5 to 6 minims), ethyl iodid (10 to 20 minims), or a few whiffs of chloroform. If such measures fail to afford relief, internal remedies must be used. In some cases strong hot coffee acts most happily; in others more benefit is derived from hot whisky and water. Among the numerous special remedies that have been advocated the following appear to be the most reliable: opium, belladonna, bromids, chloral, paraldehyd, Hoffmann's anodyne, lobelia, and quebracho.

Few attacks will resist the action of morphin hypodermically with atropin, but the greatest caution must be exercised in order that the patient may not become addicted to the drug. Heroin hydrochlorid hypodermically, in doses of from $\frac{1}{12}$ to $\frac{1}{10}$ grain, may often be substituted for morphin with great advantage. When the attacks are associated with bronchial catarrh, a combination like the following sometimes proves efficacious:

℞.	Tincturæ belladonnæ	fʒj
	Tincturæ lobeliæ	fʒiij
	Extracti aspidospermatis fluidi	f℥ss
	Spiritus ætheris compositi	fʒv
	Strontii bromidi	ʒiiss
	Elixiris aromatici . . . . . . q. s. ad	f℥iv.—M.

SIG.—A dessertspoonful in water every two or three hours.

Among other measures that have been found useful in alleviating asthmatic attacks may be mentioned the application of sinapisms to the chest, the inhalation of compressed air, and the inhalation of oxygen.

HAY ASTHMA.

(Hay-fever; Autumnal Catarrh; Rose-cold.)

Definition.—A catarrhal affection of the upper air-passages, characterized by coryza and asthmatic seizures, and evoked by irritation of a hyperesthetic nasal mucous membrane.

Etiology.—An inherited tendency, nervous temperament, indoor life, and chronic nasal disease are predisposing factors. The attacks, as a rule, occur in the autumn (autumnal catarrh) or in the spring (rose-cold), and are excited by certain dusts, vapors, or odors. The pollen of plants seems to be a common excitant. The seizures may occur at any time if the peculiar irritant is present.

Pathology.—An essential feature is the hypersensitive condition of the mucous membrane, and this is often, though not invariably, associated with hypertrophic rhinitis.

Symptoms.—Redness of the conjunctivæ and swelling of the eyelids; pruritus of the nose and eyes; sneezing; obstruction of the nostrils; watering of the eyes; a copious discharge of mucus from the nose; headache; cough; and asthmatic attacks are the usual phenomena.

Rose-cold usually begins in May or June and runs to the latter part of July. *Autumnal catarrh* begins in the latter part of August and ends with the first frost.

Prognosis.—The disease never proves fatal, but permanent cure is very rare.

Treatment.—Careful search should be made for chronic nasal disease, and if found, appropriate treatment instituted.

A change of climate during the period of susceptibility exempts most patients. A sea-voyage or a sojourn in some high-mountain district, like the White Mountains, Adirondacks, Catskills, or Alleghanies, may be recommended.

Tonics such as quinin, arsenic, and strychnin are often very useful when administered before and during an attack. To allay itching and lacrimation the eyes may be washed with a solution of boric acid (10 grains to the ounce) or sulphate of zinc (1 to 2 grains to the ounce). Sneezing, nasal fulness, and discharge are often relieved by medicated sprays (Dobell's solution) or the application, on pledgets of cotton, of adrenalin solution (1 : 5000). In some cases Dunbar's pollantin acts very favorably.

PULMONARY EMPHYSEMA.

Definition.—Abnormal distention of the lungs with air.

Varieties.—(1) **Interlobular Emphysema.**—This form is rare, and results from the rupture of the air-vesicles and the escape of air into the interstitial tissue.

(2) **Compensatory Emphysema.**—This is a vicarious distention of one part of the lung, owing to pathologic changes in another part of the organ. It is primarily physiologic,

though atrophy of the walls of the air-vesicles may ultimately ensue.

(3) **Atrophic or Senile Emphysema.**—In this form the capacity of the air-vesicles is relatively increased, owing to atrophy of the solid tissue.

(4) **Hypertrophic or Substantive Emphysema.**—This is the ordinary form of emphysema. It is characterized by a great enlargement of the lungs in consequence of overdistention of the air-vesicles.

Compensatory emphysema, atrophic emphysema, and hypertrophic emphysema together form a subdivision known as vesicular emphysema.

HYPERTROPHIC EMPHYSEMA.

Definition.—A pulmonary disease characterized anatomically by dilatation of the air-vesicles and atrophy of their walls; and clinically by permanent enlargement of the thorax, with persistent dyspnea.

Etiology.—Congenital weakness of the lung structure—probably a defective development of elastic tissue—is an important predisposing factor. This predisposition may be transmitted through several generations.

In forced expiration the air cannot escape with sufficient rapidity through the narrow glottis, and the backward pressure stretches the air-vesicles; hence the obstinate cough of chronic bronchitis, the expiratory straining of asthma, and occupations that necessitate forced expiration, like playing on wind-instruments and glass-blowing, are causal factors.

Pathology.—The lungs are enlarged and do not collapse when the thorax is opened. In bad cases the free margins are studded with large bullæ or blebs that have resulted from the rupture of a number of vesicles into a common sac. The organs are pale and have a soft, cotton-like feel. Microscopic examination reveals atrophy of the vesicular walls, a diminished amount of elastic tissue, and more or less obliteration of the pulmonary capillaries. This last condition leads to increased tension in the pulmonary artery and to secondary hypertrophy of the right ventricle.

Symptoms.—The disease generally manifests itself in middle life, but it is sometimes observed in the young. Dyspnea, increased by exertion; cyanosis, often extreme during attacks of acute bronchitis; and cough, from the associated bronchitis, are the usual symptoms. In advanced cases edema of the feet may result from cardiac failure.

Physical Signs.—The neck is short, and the sternocleidomastoids are prominent. The thorax is likewise short, but broad especially in its anteroposterior diameter. This configuration has given rise to the term "barrel-shaped" chest. On respiration there is little expansion, but an elevation of the thorax as a whole. The apex-beat is invisible, but an abnormal pulsation is often noted in the epigastrium.

Palpation.—Vocal fremitus is diminished.

Percussion.—This gives increased resonance. The upper level of hepatic dulness is depressed, and the area of cardiac dulness may be almost obliterated.

Auscultation.—Inspiration is short and feeble; expiration is prolonged and low pitched, or inaudible. Râles resulting from the associated bronchitis are frequently heard. The pulmonary second sound is accentuated.

Complications.—Bronchitis, asthma, dilatation of the right ventricle, and, later, tricuspid regurgitation and dropsy.

Diagnosis.—**Chronic Bronchitis.**—In this disease there is no marked dyspnea; the chest is not enlarged; there is no change in the percussion-note or in the expiratory sound.

Pneumothorax.—This disease develops suddenly, is unilateral, and yields a tympanitic note on percussion and metallic tinkling and bell-tympany on auscultation.

Prognosis.—Emphysema is incurable, but its advance may be stayed by relieving the primary condition. It runs a long course and is in itself rarely fatal, but death may result from heart-failure and dropsy, or from intercurrent pneumonia.

Treatment.—The treatment advocated in chronic bronchitis and asthma is often applicable here. The patient should be placed under the most favorable hygienic conditions. Iron is indicated in the anemic. Strychnin ($\frac{1}{40}$ to $\frac{1}{30}$ grain) is a valuable respiratory and cardiac stimulant, and may be combined with digitalis when there are symptoms of heart-failure.

℞. Strychninæ sulphatis . . . . gr. ss
Pulveris digitalis
Pulveris scillæ
Ferri reducti . . . . āā gr. xx.—M.
Fiant pilulæ No. xx.
SIG.—One thrice daily.

The inhalation of oxygen, or the inspiration of compressed air, followed by expiration into rarefied air, is sometimes a useful measure.

HEMOPTYSIS.

(Bronchorrhagia; Bronchopulmonary Hemorrhage.)

Definition.—The expectoration of blood.

Etiology.—The chief causes are: (1) Traumatism. (2) Certain organic diseases of the lung, especially tuberculosis, lobar pneumonia, bronchiectasis, gangrene, infarct, and cancer. (3) Passive congestion the result of heart disease, especially mitral lesions. (4) Rupture of an aortic aneurysm. (5) Diseases profoundly affecting the blood, such as purpura, hemophilia, scurvy, and leukemia. (6) Ulcers, traumatic, syphilitic, or malignant, of the trachea or larynx. (7) Vicarious menstruation (very rare).

Symptoms.—Sometimes the bleeding is preceded by cough, dyspnea, or substernal warmth or tenderness, but often there is no premonition, and the first indication is the presence of a warm salty fluid in the mouth. The blood is generally raised by coughing, and is bright red and frothy. It is alkaline in reaction, and intimately mixed with air and mucus. The hemorrhage is rarely profuse unless it results from the rupture of an aortic aneurysm or the ulceration of a large vessel in advanced phthisis. Auscultation of the chest reveals bubbling râles. The subsequent expectorations are tinged with blood, and if much is swallowed, it may excite vomiting or pass into the intestine and impart a tarry appearance to the stools.

Diagnosis.—The differential diagnosis between *hematemesis* and hemoptysis has been considered on page 63.

Prognosis.—This depends upon the cause. It is rarely fatal, except in aneurysm and in advanced phthisis with a large cavity.

Treatment.—Absolute rest is essential. An ice-bag may be placed over the suspected seat of the hemorrhage, but it should be removed if it aggravates the coughing. Bits of ice may be given to the patient to suck. There is no more useful remedy than morphin, which serves to allay excitement and to check cough. It is best given hypodermically. The application of firm ligatures to the limbs may prove efficacious by lowering the intrapulmonary pressure. When the hemorrhage is protracted, a saline purge is sometimes useful. Among other remedies that seem to be of service may be mentioned oil of erigeron, fluid extract of hamamelis, and gelatin. Ergot is useless, and so is the inhalation of vaporized solutions of astringent drugs.

HEMORRHAGIC INFARCT OF THE LUNG.

(Pulmonary Apoplexy.)

Definition.—A circumscribed area of necrosis infiltrated with blood.

Etiology.—The most common cause of pulmonary infarct is obstruction of a branch of the pulmonary artery by an embolus coming from the right heart or the general venous system. In some cases the obstruction is caused by a thrombus, the formation of which has been favored by cardiac weakness.

Pathology.—The infarct is usually located in the periphery of the lung; it is conic in shape, with its apex pointing inward. The portion affected is firm, airless, and of a dark-red color. Microscopic examination shows a dense aggregation of blood-corpuscles.

If the process lasts long enough, the dead tissue and blood are slowly absorbed and replaced by a cicatrix.

Symptoms.—When the infarct is large, the usual symptoms are dyspnea, cough, and the expectoration of dark blood containing few air-bubbles. These symptoms occurring in chronic heart-disease are especially suggestive.

Physical Signs.—Very large infarcts may give dulness and bronchial breathing.

Treatment.—The condition itself is not amenable to treatment. Remedies should be directed to the primary disease.

CONGESTION OF THE LUNGS.

ACTIVE CONGESTION.

Etiology.—This results from an increased afflux of blood to the lungs. Violent exercise, mountain climbing, and the inhalation of irritants may produce it. It is an associated condition in all severe inflammatory diseases of the lungs. In the vast majority of cases it marks the initial stage of croupous pneumonia.

Pathology.—The lung is bright red in color, heavy, and less crepitant. When incised and pressed, copious frothy blood exudes.

Symptoms.—The chief symptoms are dyspnea; a short, dry cough, followed by frothy, blood-streaked sputum; and a rapid, full pulse. The presence of fever indicates commencing pneumonia.

Physical examination reveals slight dulness, crepitant râles, and bronchovesicular breathing.

Treatment.—The measures most likely to effect depletion of the lung are complete rest, the application of dry or wet cups to the chest, and the administration of veratrum viride and a saline purge.

PASSIVE CONGESTION.

Etiology.—This results from obstruction to the flow of blood from the lungs to the heart. The chief cause is cardiac disease, especially lesions of the mitral valves and weakness of the left ventricle from fatty or fibroid changes.

Pathology.—The lungs are dark red in color, and often somewhat edematous. When the condition has lasted a long time, the organs become brown, dense, and tough (*brown induration*). Microscopic examination reveals dilatation of the capillaries, overgrowth of connective tissue, brown pigmentation, and degenerative changes in the blood-vessels.

Symptoms.—Dyspnea, cough, and the expectoration of blood-stained mucus containing pigmented epithelial cells are the characteristic symptoms. *Physical examination* reveals slight dulness, feeble breathing, and abundant fine râles.

Treatment.—Remedies should be directed to the underlying cardiac disease. The application of dry or wet cups often gives temporary relief. When the symptoms are urgent, venesection is indicated. Saline and mercurial aperients are of service.

HYPOSTATIC CONGESTION.

(Hypostatic Pneumonia; Splenization of the Lung.)

Definition.—A congestion of dependent portions of the lungs occurring in asthenic diseases that necessitate a protracted recumbent position.

Etiology.—It is generally observed in low fevers and in chronic wasting diseases. Cardiac weakness, recumbent posture, and alterations in the blood are the causal factors.

Pathology.—The lungs are dark red and edematous posteriorly. The edema and increased amount of blood render the organs more solid and less crepitant. They never show the granular appearance of croupous pneumonia.

Symptoms.—The symptoms are often indefinite. There may be moderate dyspnea, slight cyanosis, cough, and, perhaps, blood-tinged expectoration.

Physical examination reveals dulness over the lower lobes, subcrepitant râles, and feeble, blowing breathing.

Treatment.—Efforts should be made to prevent the development of hypostatic pneumonia in asthenic diseases by frequent change of posture and the timely administration of such stimulants as strychnin, digitalis, alcohol, ammonia, and camphor. When already present, relief is sometimes afforded by dry or wet cupping or the application of stupes.

EDEMA OF THE LUNGS.

Definition.—An effusion of serous fluid into the air-vesicles and interstitial tissue of the lungs.

Etiology.—(1) It is frequently caused by passive hyperemia, the result of chronic heart disease. (2) It may be a part of a general dropsy induced by Bright's disease. (3) It is a common cause of death in conditions that lead to heart-failure, such as grave anemia, cerebral lesions, and acute infections.

Local pulmonary edema is often found around abscesses, infarcts, and areas of consolidation.

Pathology.—The lungs, especially the dependent portions, are heavy, red in color, and boggy to the feel. When the affected portion is incised and subjected to pressure, abundant blood-stained, frothy serum exudes.

Symptoms.—These consist in dyspnea, cyanosis, cough, and the expectoration of large quantities of frothy, serous fluid. Occasionally the sputum is blood-stained. The skin is often cold and livid. There is no fever.

Physical examination reveals feeble tactile fremitus, dulness, weak breath-sounds, and numerous fine, moist râles.

Diagnosis.—**Croupous Pneumonia.**—This is characterized by a chill, fever, pain, rusty expectoration, and signs of consolidation.

Hydrothorax.—In this condition there may be enlargement of the affected side, with displacement of the apex-beat. The upper level of dulness is often movable, and frothy sputum and râles are absent.

Prognosis.—Always grave. It is often a terminal symptom of the disease in which it occurs. When not far advanced, and the primary disease is amenable to treatment, recovery may follow.

Treatment.—When there is much cyanosis and the patient's strength will permit it, the application of wet cups to the chest or bleeding from the arm is of great value. Hot fomentations should be applied to the chest. Hydragogue cathartics are indicated. Epsom salts in concentrated solution or elaterium ($\frac{1}{8}$ grain) may be selected.

Cardiac stimulants like alcohol, ammonia, camphor, digitalis, and especially strychnin are required and may be given hypodermically.

Caffein is a useful diuretic and cardiac and respiratory stimulant.

CROUPOUS PNEUMONIA.

(**Lobar Pneumonia; Pneumonitis; Lung Fever.**)

Definition.—An acute specific disease, characterized anatomically by an inflammation of the lungs, followed by a rapid infiltration of their alveoli, and manifested clinically by high fever, cough, dyspnea, "rusty" sputum, and physical signs indicative of consolidation.

Etiology.—Age, sex, and climate exert but little predisposing influence. Lowered vitality from bad hygienic surroundings or from some preëxistent disease, like diabetes, Bright's disease, or one of the infectious fevers, favors its development. One attack renders the patient more liable to subsequent infection. Alcoholism is a strong predisposing factor. Exposure to cold and wet often precipitates the attack.

The exciting cause is the invasion of the lung by pathogenic bacteria, especially by Fränkel's Diplococcus pneumoniæ.

Pathology.—Anatomically three stages have been recognized: (1) That of congestion; (2) that of red hepatization; (3) that of gray hepatization.

Stage 1.—The affected portion remains distended when the chest is opened; it is of a deep-red color, and is more resistant to the touch than the normal lung. On section, a frothy, blood-stained serum freely exudes. Microscopic examination reveals a dilated and tortuous condition of the capillaries, swelling of the alveolar cells, and a slight corpuscular exudate.

Stage 2.—The hepatized portion is increased in volume, is quite firm, is of a dark-red color, and so heavy that it sinks in water. It is very friable, and the torn surface is dry and presents a granular appearance, owing to the projection of fibrinous plugs from the alveoli.

Microscopic examination reveals a mesh of coagulated fibrin inclosing numerous red blood-corpuscles and some leukocytes. The latter are also noted in the interlobular tissue. In sections properly treated the diplococcus is detected.

Stage 3.—The red color gives place to a mottled gray, and the solidified area begins to soften. The change in color is due to the compression of the capillaries, to the disappearance of red corpuscles and their replacement by leukocytes, and to fatty degeneration of some of the elements.

The consolidation usually begins at the base and extends upward. The most frequent seat is the lower lobe of the right lung. The bronchi and the adjacent pleura are involved in the inflammatory process.

Events.—Resolution commonly occurs, the exudate being removed rapidly by absorption. Death may occur at any period of the disease from general toxemia, the severity of which is often altogether disproportionate to the area of lung involved; from dilatation of the right ventricle; from asphyxia; or a pneumococcic complication, such as meningitis or endocarditis.

Abscess, gangrene, and chronic interstitial pneumonia are rare terminations.

Symptoms.—The disease usually begins with a decided *chill* and a sharp *pain* in the side, followed by a rapid *rise of temperature*. The latter often attains its maximum (104°–105° F.) in twenty-four hours, and generally continues high, with slight diurnal remissions, until the fifth, seventh, ninth, or eleventh day, when it falls by crisis, frequently reaching the norm within twenty-four hours. Occasionally the temperature falls by lysis. There is marked *dyspnea;* the respirations are shallow and rapid, ranging from 40 to 80 a minute, thus making the ratio between the respiration and the pulse as 1 is to 3 or as 1 is to 2. *Cough* is a prominent symptom: at first it is short and dry, but later it is accompanied by *bloody or rusty translucent and tenacious sputa.* Microscopically the sputum contains red blood-corpuscles, free pigment, pus-corpuscles, diplococci, and other micro-organisms. The face is flushed; the lips are cyanosed and often the seat of a herpetic eruption; the tongue is heavily furred; the bowels are constipated; the urine is scanty, high-colored, deficient in chlorids, and often slightly albuminous. In severe cases *delirium* is rarely absent. Examination of the blood usually shows marked leukocytosis.

Physical Signs.—*Inspection.*—There may be deficient expansion over the affected side. There is no bulging of the interspaces nor displacement of the apex-beat.

Palpation.—In the vast majority of cases the vocal fremitus is considerably increased over the affected area.

Percussion.—In the earliest stage there may be hyperresonance from diminished intrapulmonary tension. As consolidation advances, however, the note becomes remarkably dull. Percussion over unaffected lobes yields hyperresonance or tympany.

Auscultation.—In the stages of congestion fine crepitant râles are heard at the end of full inspiration. They are probably produced by the forcible separation of adherent vesicular walls. In the stage of consolidation auscultation reveals exaggerated vocal resonance and bronchial breathing. During resolution the softening of the exudate gives rise to fine moist râles—the *redux crepitus.*

Atypical Cases.—*Senile Pneumonia.*—The symptoms often develop insidiously; the temperature may not be high; the pulse may not be accelerated; expectoration is often absent; the signs are not marked; delirium is common; weakness is extreme; and death from exhaustion is the most frequent termination.

Pneumonia in Children.—It is often ushered in with convulsions. Headache, delirium, stupor, and coma are prominent symptoms, so that the disease may simulate meningitis. The temperature is very high; expectoration is often absent. The disease frequently begins at the apex of the lung.

Typhoid Pneumonia.—In this form there are pronounced typhoid symptoms—headache, muttering delirium, stupor, a dry, brown tongue, subsultus tendinum, carphologia, a rapid, weak pulse, and high fever. The expectoration may resemble prune-juice.

Pneumonia of Drunkards.—The onset is often gradual; the dyspnea is marked; the temperature is not high; violent maniacal delirium commonly develops; and death from exhaustion is exceedingly frequent.

Massive Pneumonia.—In this form the bronchi, as well as the air-vesicles, are filled with fibrinous exudate. The physical signs resemble those of pleural effusion.

Central Pneumonia.—In this form the inflammatory process commences in the center of a lobe, and in consequence the characteristic physical signs may not manifest themselves for two or three days.

Migratory Pneumonia.—In this type the specific inflammation shows a tendency to spread and to involve successively fresh areas of lung tissue.

Complications.—These are usually due to pneumococcic infection. Pleurisy is the most common complication. It may be either serous or purulent. Pericarditis and endocarditis are not very infrequent. The latter is often ulcerative in type. Among less frequent complications may be mentioned meningitis, arthritis, parotitis, nephritis, jaundice, and delayed resolution (consolidation may last for five or six weeks and then gradually disappear). Abscess, gangrene, and chronic interstitial pneumonia are rare sequels.

Diagnosis.—**Pleurisy.**—There is rarely a distinct chill; fever is not so high nor the pulse so rapid; there is no rusty sputum; nervous symptoms are wanting; there is often bulging of the interspaces, with displacement of the apex-beat; the level of dulness may change with the posture of the patient; vocal fremitus and vocal resonance are diminished; and the breath-sounds are generally weak and distant.

Acute Phthisis.—The history, the mode of onset, the long duration, the remittent fever, the rapid emaciation, profuse sweats, and presence of tubercle bacilli and elastic fibers in the sputum will suggest phthisis.

Pulmonary Edema.—In edema there is absence of chill, fever, and pain; the expectoration is frothy and serous; both lungs are commonly affected; auscultation reveals abundant subcrepitant râles and weak breathing.

Typhoid Fever.—Typhoid pneumonia may readily be mistaken for typhoid fever with pneumonia; but pneumonia as a complication occurs late in the disease, so that the history of the onset gives much assistance.

Prognosis.—In young, robust subjects of good habits the prognosis is good. After the age of sixty the outlook is grave. In drunkards the disease is especially fatal. The coexistence of heart or kidney disease makes pneumonia exceedingly dangerous.

In individual cases continued high fever (above 103.5° F.), a pulse more rapid than 120 a minute, severe nervous symptoms, extensive consolidation, and absence of leukocytosis are unfavorable factors. The average mortality is about 20 per cent.

Treatment.—The temperature of the sick-room should be between 65° and 70° F. The diet should be fluid or semifluid. Milk, junket, wine-whey, broths, eggs, and gruel are suitable forms of nourishment. Cool water should be given freely. In the absence of any indication for special local treatment the chest may be enveloped in a cotton jacket.

In robust subjects, at the very onset, when the invasion is violent and attended with a bounding pulse, marked dyspnea, and severe pleuritic pain, the abstraction of from 10 to 20 ounces of blood may afford great relief. Later in the course of the disease, if cyanosis and orthopnea develop in consequence of overdistention of the right ventricle, venesection may also prove useful.

Cardiac Weakness.—Alcohol is the best stimulant. When the pulse becomes compressible and the diastolic sound at the pulmonary area loses its force, it should be given freely. The patients who need it most are the old, the debilitated, and the alcoholic. Digitalis is undoubtedly useful in some cases, but its action is uncertain and often disappointing.

As a circulatory stimulant strychnin generally proves much more efficacious than digitalis. It should be given in ascending doses of from $\frac{1}{60}$ to $\frac{1}{15}$ of a grain. In order that there may be immediate absorption, large doses should always be given hypodermically. Caffein is a useful adjuvant to strychnin, but it should not be used when there is marked insomnia. In threatening collapse camphor hypodermically (1 to 2 grains in sterile olive oil every two or three hours) is very efficient. Subcutaneous injections of normal salt solution have also been found useful in overcoming adynamia.

Pain.—Morphin hypodermically is the best analgesic. Hot or cold applications are useful. When the pain is very severe, a few wet cups, followed by poultices, will be found serviceable.

Cough.—Hard, dry cough is best relieved by codein ($\frac{1}{8}$ to $\frac{1}{4}$ grain), heroin ($\frac{1}{16}$ grain), or Dover's powder (3 to 5 grains). Expectorants are rarely needed. When, however, there is much bronchial catarrh, ammonium carbonate may be given to facilitate expectoration.

Fever.—Persistent high fever is best controlled by the application of ice-bags to the affected side, cold sponging, or cool baths.

Dyspnea.—Cardiac and respiratory stimulants (strychnin, caffein, ammonia) are of service. Oxygen makes the breathing easier, lessens cyanosis, and conduces to sleep, and to this extent aids in conserving energy.

Insomnia and Delirium.—Opium is generally the best sedative. Of course it should not be used when there is extreme dyspnea or when there are evidences of pulmonary edema. Bromids or chloralamid may be tried.

Delayed Resolution.—Small blisters may be applied over the affected area, and potassium iodid may be administered internally.

℞. Ammonii iodidi . . . . ʒj
Ammonii chloridi . . . . ℥iss
Misturæ glycyrrhizæ compositæ . . . . f℥vj.—M.
SIG.—A tablespoonful in water four times a day.

CATARRHAL PNEUMONIA.

(Capillary Bronchitis; Bronchopneumonia; Lobular Pneumonia.)

Definition.—An inflammation of the terminal bronchioles and air-vesicles.

Etiology.—It is most frequently observed in the very young and the old. It is a common sequel of the specific fevers, especially of whooping-cough, measles, influenza, and diphtheria. In debilitated subjects it may occur as a primary affection, the result of exposure.

Another group of cases results from the aspiration of particles of food into the smaller bronchi (aspiration or deglutition pneumonia). This accident is liable to occur whenever the sensibility of the larynx is benumbed, as in apoplexy, bulbar palsy, or uremia. Cancer of the throat and operations on the upper air-passages also favor its occurrence.

The immediate cause is some bacterium. The organism most frequently found is the Diplococcus pneumoniæ. This may occur alone or in combination with the streptococcus, staphylococcus, bacillus of Friedländer, or more rarely with the typhoid bacillus, influenza bacillus, colon bacillus, or diphtheria bacillus.

Pathology.—As a rule, both lungs are involved. On section, small projecting areas of consolidation are noted here and there around the finer bronchioles. Recent patches are reddish-brown in color, firm, and smooth or finely granular; later they become grayish and soft. The terminal bronchi are filled with purulent material.

In addition to these solidified areas there are other small patches of collapsed lung that are airless, firm, and bluish-red in color. The collapse has resulted from occlusion of the bronchus, and closely resembles consolidation; but it can, as a rule, be overcome when inflation is practised by means of a blowpipe inserted in the supplying bronchus.

Microscopic examination reveals an exudate in the terminal bronchi and air-cells, which is composed of leukocytes and desquamated epithelium in various stages of degeneration. The walls of the bronchioles are also infiltrated with leukocytes.

When compared with croupous pneumonia, the contrast is striking. In the latter the lung is involved *en masse*; the consolidation is distinctly granular, and the exudate is composed of red blood-corpuscles, white blood-corpuscles, and fibrin; the lining epithelium is but slightly involved, and the walls of the bronchi are not infiltrated with leukocytes.

Terminations.—(1) Resolution; the exudate undergoes fatty degeneration and is eventually absorbed or expectorated. (2) Death frequently occurs from asphyxia or exhaustion. (3) Termination in tuberculosis was supposed formerly to occur very frequently; it is now regarded as being relatively rare. Most of the cases in which this termination is supposed to have occurred were in reality cases of primary tuberculous pneumonia. (4) Abscess, gangrene, and chronic interstitial pneumonia are occasional sequels.

Symptoms.—The symptoms are often masked by the primary disease. The onset is usually gradual, and is characterized by prostration, cough, and fever. The last is moderately high and very irregular (101°–104° F.). The dyspnea is marked, and the respirations are rapid—50 to 80 a minute; the pulse is greatly accelerated—120 to 180 a minute; cough is painful and accompanied by a mucopurulent expectoration that is rarely blood-streaked. The face is usually pale and anxious, and the lips blue.

Physical Signs.—As the areas of consolidation are generally small and scattered, the physical signs are not marked.

Inspection reveals evidences of dyspnea—lividity, playing of the nostrils, prominence of the sternocleidomastoids, and perhaps retraction of the base of the chest.

Palpation usually gives negative results.

Percussion may reveal areas of dulness in one or both lungs.

Auscultation reveals whistling and subcrepitant râles and areas over which the breathing is bronchial or bronchovesicular.

Diagnosis.—**Acute Phthisis.**—In this disease there is a tuberculous bronchopneumonia which is difficult to distinguish from simple bronchopneumonia. A family history of tuberculosis, extensive involvement of the apices, bubbling râles indicating softening, the long duration, profuse sweats, rapid emaciation, and the presence of tubercle bacilli and elastic fibers in the sputa are the diagnostic phenomena of phthisis.

The following table will show the clinical differences between *catarrhal* and *croupous pneumonia:*

CATARRHAL PNEUMONIA.	CROUPOUS PNEUMONIA.
Usually secondary to bronchitis or an acute infectious disease.	Usually a primary disease.
The onset is gradual and without a distinct chill.	The onset is abrupt and with a distinct chill.
The fever is moderately high, very irregular, and ends by lysis after an indefinite period, sometimes of two or three weeks' duration.	The fever is high, regular, and generally ends by crisis between the sixth and ninth day.
The sputum is mucopurulent or glairy and tenacious.	The sputum is rusty and translucent.
Both lungs are commonly affected.	In the majority of cases only one lung is affected.
The physical signs are indistinct and indicate scattered areas of consolidation.	The physical signs are distinct and indicate a large uniform consolidation.

Bronchitis.—In simple bronchitis the fever is not high; the dyspnea is slight, there is little prostration, and there are no signs of consolidation.

Prognosis.—In previously healthy children the prognosis is good. In cachectic children the outlook is very grave. Aspiration pneumonia is generally fatal. The average mortality is about 35 per cent.

The duration of the disease is from one to three weeks; a longer duration should suggest tuberculosis.

Treatment.—Much can be done by careful management in preventing bronchitis from gaining access to the smaller bronchi.

On the supervention of catarrhal pneumonia the patient should be confined to bed, and the temperature of the room maintained between 68° and 70° F. The atmosphere should be rendered moist with steam. The diet should be liquid and nutritious.

Alcohol is often required. When the circulatory depression is pronounced, whisky may be given in doses of from 10 to 30 minims in milk to a child of two years every two or three hours.

At the outset it is advantageous to administer a mild purgative, preferably calomel or castor oil. A jacket of cotton-wool should be worn throughout the attack. When there is a harsh, dry cough, the application of the tincture of iodin of suitable strength generally affords some relief. In adults sinapisms or stupes may be used instead of the iodin.

Fever is best controlled by cold. Compresses wrung out of cold water may be wrapped around the chest and changed for fresh ones at intervals of twenty minutes. Expectorants are usually required. In the early stage potassium citrate is very serviceable. It may often be combined advantageously with spirit of nitrous ether and ammonium acetate, as in the following formula:

℞. Potassii citratis . . . . ʒiss
Spiritus ætheris nitrosi . . . . f℥vj
Liquoris ammonii acetatis . . . . fʒj
Syrupi tolutani
Aquæ . . . . āā q. s. ad f℥iv.—M.
Sig.—Dessertspoonful every three hours for a child of three years.

Later, the ammonium salts, especially the carbonate, are more efficacious. From 1 to 2 grains of the latter may be given every three or four hours to a child of two years. Ammonium iodid is also useful, and may be employed as an adjuvant, as in the following formula:

℞. Ammonii carbonatis . . . . gr. xlviij
Ammonii iodidi . . . . gr. xxiv
Syrupi tolutani
Syrupi acaciæ . . . . āā q. s. ad f℥iij.—M.
Sig.—Teaspoonful every two or three hours for a child of three years.

When the child is unable to expel the mucus and the breathing becomes much oppressed, an emetic (ipecac or alum) may prove of great service. Inhalations of oxygen sometimes make breathing easier. Strychnin is also of benefit at this time in combating respiratory failure.

If symptoms of cardiac failure are pronounced, digitalis must be given in addition to alcohol and strychnin. Extreme restlessness and insomnia will sometimes require the use of the bromids or some other mild sedative.

Except at the onset, when they may be necessary to relieve pleuritic pain and to control harassing cough, opiates should not be used.

Convalescence must be guarded. Tonics like cod-liver oil, iron, and hypophosphites are useful restoratives. A change of air is desirable in protracted cases.

CHRONIC INTERSTITIAL PNEUMONIA.

(**Cirrhosis of the Lung; Chronic Pneumonia; Pulmonary Induration.**)

Definition.—A chronic disease of the lung, characterized by an overgrowth of fibrous tissue.

Etiology.—It is a rare sequel of croupous or catarrhal pneumonia. It may be excited by the constant inhalation of irritating dusts, as stone-dust (chalicosis), coal-dust (anthracosis), or metal-dust (siderosis). It may result from syphilis. It is occasionally secondary to chronic pleurisy. It is an invariable accompaniment of chronic phthisis.

Pathology.—When the thorax is opened, the lung is found retracted and the heart displaced. The organ is tough, firm, and more or less airless. Section shows an overgrowth of fibrous tissue, and usually inflammation and considerable dilatation of the bronchi.

Symptoms.—The chief symptoms are dyspnea on exertion and cough. The latter may be dry, but it is usually associated with more or less mucopurulent sputum. There is no fever, and the general health may be well preserved for many years.

Physical Signs.—*Inspection* reveals retraction of the affected side and displacement of the apex-beat.

Percussion may yield dulness. Over saccular dilatations of the bronchi there may be a tympanitic note.

Auscultation.—The vocal resonance is increased, and the breathing is often bronchial or cavernous.

Diagnosis.—**Fibroid Phthisis.**—This is often bilateral; fever is a frequent accompaniment, and tubercle bacilli are present in the sputa.

Prognosis.—The disease is incurable. Its course, however, is extremely chronic.

Treatment.—This is largely hygienic, and coincides with that laid down for tuberculosis. Stimulant expectorants are useful when bronchitis or bronchiectasis is a prominent feature.

ABSCESS OF THE LUNG.

Etiology.—(1) It is a rare sequel of croupous or catarrhal pneumonia. (2) It is a frequent accompaniment of tuberculosis. (3) It may be excited by the inhalation of foreign bodies. (4) It may result from the extension of a suppurative inflammation in some neighboring part, such as the pleura or liver. (5) Multiple abscesses of embolic origin are of common occurrence in pyemia.

Symptoms.—High and irregular fever, rigors, sweats, pallor, and leukocytosis indicate suppuration. Dyspnea, cough, and purulent, offensive sputa containing shreds of lung tissue are the pulmonary symptoms. Physical examination may reveal bubbling râles, and, later, cavernous breathing and pectoriloquy. Multiple embolic abscesses are rarely recognized during life.

Prognosis.—Many cases following pneumonia and the rupture of external abscesses into the lung recover. Embolic abscess always prove fatal.

Treatment.—Nutritious food and quinin, strychnin, and alcoholic stimulants will be required to support the system. Single abscesses, when they can be localized, should be opened and drained.

GANGRENE OF THE LUNG.

Etiology.—Gangrene of the lung is not a primary condition, but is secondary to inflammation or necrosis of the lung tissue. It is excited by the entrance of bacteria of putrefaction, but unless the system is considerably reduced in vitality, the tissues, even though diseased, show wonderful resistance and escape putrefaction.

Pneumonia, especially aspiration-pneumonia, phthisis, pressure of morbid growths, bronchiectasis, abscess, and hemorrhagic infarction following embolism of the pulmonary artery are the predisposing pulmonary conditions; Bright's disease, alcoholism, the infectious fevers, and particularly diabetes, by lowering vitality, render the lung more liable to be attacked.

Pathology.—The process may be circumscribed or diffuse. The affected part is converted into a greenish-black, soft mass having an extremely fetid odor. When the softened material has been expectorated, there is left behind a cavity with ragged walls, containing a foul-smelling liquid. The tissues around the cavity are inflamed and edematous.

Symptoms.—Persistent cough, irregular fever, and emaciation are usually present. Hemoptysis is a frequent occurrence. The expectoration is characteristic; it is profuse, and has a penetrating, offensive odor. When allowed to stand in a glass vessel, it separates into three layers: a frothy layer on top, a translucent serous layer in the middle, through which hang strings of pus, and at the bottom a layer of reddish-green purulent material. Altered blood may give it the appearance of prune-juice. Microscopically it contains shreds of tissue, crystals of fatty acids, crystals of hematoidin, and numerous pyogenic bacteria.

Physical examination may reveal bubbling râles, and later cavernous breathing, pectoriloquy, and localized tympany on percussion. Physical signs of pyopneumothorax may supervene from perforation into the pleura.

Prognosis.—Grave, but not hopeless. Quite a number of cures have been recorded. Death may result from exhaustion, hemorrhage, or cerebral abscess the result of embolism.

Treatment.—Nutritious food and strychnin, quinin, and alcohol are required to support the system. Inhalations of creasote or of formalin (2 per cent. gradually increased to 5 per cent.) may be employed to lessen the fetor of the breath. Surgical interference is indicated when the gangrenous process can be localized and is not a complication of an incurable disease.

PULMONARY TUBERCULOSIS.

(**Phthisis; Pulmonary Consumption.**)

Definition.—A specific inflammatory disease of the lungs caused by the Bacillus tuberculosis; characterized anatomically by a cellular infiltration that subsequently caseates, softens, and leads to ulceration of the lung tissue; and manifested clinically by wasting, exhaustion, fever, and cough.

Etiology.—It most commonly develops between the ages of fifteen and forty. (1) An inherited susceptibility to infection; (2) residence in low, damp, and badly drained localities; (3) occupations that necessitate the breathing of impure air and irritating dusts; (4) catarrhal affections of the respiratory tract; (5) chronic alcoholism; (6) certain general diseases, such as whooping-cough, measles, diabetes, cirrhosis of the liver, and nephritis are important predisposing factors.

The exciting cause is the Bacillus tuberculosis. Infection takes place—(1) By the inhalation of air laden with the dust of dried tuberculous expectoration; (2) by the ingestion of tuberculous milk or meat; (3) by the direct inoculation of wounds (rare); and (4) by direct parental transmission (very rare).

Pathology.—The Bacillus tuberculosis is a very minute rod, about one-fourth or one-half as long as the diameter of a red blood-corpuscle, and often slightly bent and beaded. Its detection depends on the power of the stained bacillus to resist the decolorizing effects of acids. (See p. 204.)

The lodgment of the bacilli in the terminal bronchioles or peribronchial tissues excites a proliferation of the fixed connective-tissue cells. The new cells, from their resemblance to epithelial cells, are known as *epithelioid cells*. They have a relatively large amount of protoplasm and a rather faintly staining nucleus. *Giant-cells* are sometimes formed by the fusion or overgrowth of the epithelioid cells. In consequence of the local irritation the cellular proliferation is soon surrounded by a wall of *leukocytes*, the whole forming a gray, translucent mass—the gray tubercle of Laennec. In a short time the bacilli excite a coagulation-necrosis that starts in the center, spreads to the periphery, and converts the tubercle into a yellow, cheesy mass—the yellow tubercle of Laennec. The degenerated tubercles fuse and form the uniform cheesy masses so commonly observed at the autopsy. At this stage one of two things may occur: the mass may soften, break into a bronchial tube, and leave behind a cavity with ulcerating walls, or it may become encapsulated by an overgrowth of connective tissue and subsequently calcified. In addition to the specific process other secondary changes are noted. The lung tissue in the neighborhood of the tuberculous deposits is often the seat of a true pneumonic inflammation; the connective tissue is always more or less proliferated; the bronchial tubes are inflamed; and the pleural surfaces over the affected areas are nearly always adherent.

Chronic ulcerative phthisis usually begins at the apices.

Acute phthisis has been termed *phthisis florida*, *cheesy pneumonia*, and *chronic catarrhal pneumonia*, but the process is invariably tuberculous. From extreme vulnerability of the tissues a lobe or whole lung, or even both lungs, is rapidly infiltrated, and death results in from a few weeks to a few months.

In some cases the lung is solidified by a dense, yellowish-gray infiltration composed of closely aggregated tubercles; in others the consolidation appears in more or less discrete patches that have had their origin in the smaller bronchial tubes; in a third form one or both lungs are studded with discrete tubercles, many of which are still gray and translucent.

In *fibroid phthisis* the tissues are more resistant, and in consequence the process is limited by an overgrowth of connective tissue that forms dense bands around the tuberculous foci. This form lasts many years.

Chronic Ulcerative Phthisis.— **Symptoms.**—The onset is usually insidious and marked by pallor, gastric disturbance, loss of flesh and strength, and by a dry, hacking cough that is noted especially in the morning. From some undue exposure the cough is often aggravated, and to this obstinate " cold " the disease is usually attributed. In some cases the symptoms appear abruptly with hemorrhage or an acute pleurisy.

Slight fever and acceleration of the pulse are early symptoms of great diagnostic import. The temperature is marked by an evening exacerbation, during which the face is flushed, the eyes are bright, and the mind animated. As the disease advances the cough becomes troublesome and the expectoration more abundant. In well-developed cases the expectoration is greenish in color, is in coin-shaped plugs (nummular), is heavy and sinks in water, is often blood-streaked, and on microscopic examination is found to contain bacilli and fibers of elastic tissue.

Phthisis is in itself not a painful disease, but the associated dry pleurisy often causes much suffering. Hemoptysis occurs at all stages, but the profuse hemorrhages occur late. The blood is bright red in color, frothy, and mixed with mucus. Dyspnea is rare until the disease is far advanced. Profuse sweating during sleep is a troublesome feature of advanced phthisis.

The final stage is characterized by extreme emaciation, weakness, pallor, high remittent or intermittent fever, and edema of the feet. The mind is usually clear and peculiarly hopeful to the end. The average duration is about two years.

Physical Examination.—The chest may be well formed. Often, however, it is long and flat, with hollow supraclavicular and infraclavicular spaces, prominent scapulæ, and oblique ribs. When the disease is well advanced, there may be retraction with diminished expansion over one apex.

Palpation.—This reveals imperfect expansion and exaggerated vocal fremitus.

Percussion.—Dulness can be detected at an early period of the disease. It may be obtained first above or below the clavicles, in the supraspinous fossæ, between the scapulæ, or in front, near the sternal border.

A cavity may yield tympany or a cracked-pot note. The latter is best obtained with quick, light percussion strokes, when the patient's mouth is open.

Auscultation.—In the earliest stage respiration may be inaudible over the affected area. Later the breathing is harsh and the expiration is prolonged (bronchial). The vocal resonance is increased. Crackling râles are usually audible, and are produced by liquid in the small tubes. If not present, coughing will usually develop them. Auscultation over cavities may detect cavernous or amphoric breathing, bronchophony or pectoriloquy, and large gurgling râles.

Anomalous Physical Signs.—The vocal fremitus is diminished when there is much pleural thickening. Normal resonance or hyperresonance may replace dulness when there is much emphysema between small tuberculous foci. Weak breathing may replace bronchial or cavernous when the tubes or cavity are filled with mucopus. The signs of cavity are sometimes produced by consolidation in the neighborhood of a large bronchus.

Acute Phthisis.—Clinically this form resembles pneumonia, and is marked by a chill, high fever, rapid pulse, dyspnea, sputum at first rusty and then purulent, flushed face, profuse sweats, and the signs of consolidation. Instead of ending by crisis at the eighth or ninth day, as in ordinary pneumonia, the symptoms gradually grow worse, signs of softening develop, bacilli and elastic fibers appear in the sputum, emaciation and anemia become pronounced, and death results in from a few weeks to a few months.

Fibroid Phthisis.—This is a disease of long duration. It is characterized by very gradual loss of flesh and strength and by an abundant mucopurulent expectoration, which is at times fetid from being retained in dilated bronchi. Dyspnea, sweating, and fever are slight. There is very marked retraction on the affected side from the shrinking of the fibrous tissue; with this exception the physical signs are similar to those of ulcerative phthisis.

Complications of Phthisis.—The chief are: hemoptysis; catarrhal pneumonia; pleurisy; pneumothorax; stomatitis; gastric catarrh; diarrhea; amyloid degeneration of the viscera; fistula in ano (tuberculous); and secondary tuberculosis of other organs, especially of the larynx, cerebral meninges, intestines, peritoneum, or kidneys.

Diagnosis.—The irregular fever, cough, pallor, emaciation, hemoptysis, night-sweats, signs of consolidation, and the presence of bacilli and elastic fibers in the sputa are the diagnostic phenomena.

Prognosis.—In acute phthisis the outlook is wholly unfavorable. In chronic phthisis the prognosis is dependent upon the stage of the disease, the constitutional vigor of the subject, and the hygienic conditions under which he is obliged to live. The accidental discovery of calcified tubercles at autopsies furnishes abundant proof of the curability of the disease. The mortality is very high in young subjects (fifteen to twenty-five years) and those of feeble constitution. Unfavorable prognostic signs are a persistent high temperature, rapid pulse (110 to 120), involvement of both lungs, continued indigestion, progressive loss of flesh, and the development of tuberculous lesions in other organs.

Treatment.—*Prophylaxis.*—Tuberculous patients should be taught to expectorate only into proper receptacles containing a disinfectant solution (5 per cent. carbolic acid) or into moistened rags or paper napkins that should be burned before the sputum becomes dry. They should sleep alone. Their rooms should be sunny, well ventilated, and kept scrupulously clean.

Much can be done by the State to limit the dissemination of the disease. Laws should be enacted providing for the systematic inspection, by skilled veterinarians, of all dairies and slaughter-houses with the view of declaring unmarketable the milk and meat of tuberculous animals.

Compulsory registration of phthisical patients is desirable. Spitting upon sidewalks and the floors of public buildings and conveyances should be made a penal offense. Finally, the State should provide special hospitals for the indigent suffering from tuberculosis.

Persons with a predisposition to tuberculosis can do much to increase their powers of resistance by strict attention to hygiene. Fresh air and sunlight, a healthy residence, an outdoor occupation, the wearing of warm clothes, with flannel next to the skin, and a diet of wholesome and nutritious food, temperate living, systematic exercise, and daily cold sponging, followed by friction of the skin, are the factors to be relied upon in attempting to overcome individual susceptibility.

Finally, all local foci of tuberculosis, such as frequently appear in the cervical lymph-glands, joints, and bones, should receive immediate attention.

Sanatorium treatment undoubtedly gives the patient the best chance of recovery. In such institutions the patient spends in summer not less than nine or ten hours, and in winter not less than from six to nine hours, in the open air. The bedroom windows are kept open both winter and summer. He is given a mixed diet of wholesome food, and encouraged to eat as heartily as his digestive capacity will permit. When the disease is active, he is kept at absolute rest. For the most of the day he lies on a bamboo couch in the open air, warmth being maintained by abundant covering and, if necessary, by a hot stone at the feet. In quiescent tuberculosis moderate exercise is recommended, every precaution being taken, however, to guard against fatigue. To secure lasting improvement, the patient should remain in the sanatorium at least six months.

Climatic Treatment.—To patients to whom a protracted stay in a sanatorium would be irksome or distasteful a change of climate offers the greatest hope of cure. As a rule, a high altitude should be selected; the atmosphere should be dry, and the temperature equable. Personal experience must decide the question of temperature; generally patients who feel better in summer will do well in a warm climate, and vice versâ. The physician should have some knowledge of the locality, which should afford ordinary conveniences without being too crowded with sufferers similarly afflicted.

In selected cases a sea voyage is often very useful. According to Douglas Powell, it is most suitable to patients in the early stages, who have been previously healthy, who have overworked nervous systems, and in whom the disease is more or less quiescent.

Treatment at Home.—This should be made to imitate as closely as circumstances will permit that which is followed in the sanatorium. The airiest and sunniest room should be selected for the patient. So long as he has fever absolute rest should be insisted upon. As much nourishing food should be allowed as he is capable of digesting.

Medicinal Treatment.—When well tolerated and digested, cod-liver oil (1 to 4 fluidrams thrice daily) is of service in improving the general nutrition. Creasote is useful when the expectoration is free and purulent. The dose should be cautiously increased from 2 or 3 minims to 15 or 20 minims, three times a day. Alcohol is useful in some cases. Malt liquors and wines are usually the best preparations. Tonics—arsenic, iron, hypophosphites—are often serviceable. Iodin appears to be effective in chronic cases. A small amount of an ointment of europhen (10 per cent.), a compound containing much loosely combined iodin, may be rubbed into the chest twice daily. Counterirritation by means of small blisters is also efficacious in chronic forms.

Symptomatic Treatment.—*Cough.*—In many cases cough is indispensable and is best treated by promoting expectoration. For this purpose creasote, guaiacol carbonate, terebene, oil of eucalyptus, and myrtol are reliable remedies. Inhalations of ipecac, creasote, compound tincture of benzoin, or terebene are often very effective. Local blistering is also of service. When the cough is very severe, sedatives must be given. Of these, the least objectionable are codein, heroin, hydrocyanic acid, and spirit of chloroform. Such combinations as the following will be found useful:

℞.	Codeinæ sulphatis	gr. vj–viij
	Spiritus chloroformi	f℥j
	Glycerin	f℥j
	Succi limonis	f℥ss
	Aquæ . . . . q. s. ad	f℥iij.—M.

SIG.—A teaspoonful as occasion demands.

℞.	Codeinæ sulphatis	gr. iv
	Acidi hydrocyanici diluti	♏ xxxij
	Syrupi tolutani . . . . q. s. ad	f℥ij.—M.

SIG.—A teaspoonful as required.

Night-sweats.—Sponging the body at bedtime with a solution of alum in alcohol and water or dusting it with a powder of tannoform (1 part) and zinc oxid (3 parts) is sometimes very effective. The most reliable internal remedies are atropin ($\frac{1}{150}$ to $\frac{1}{120}$ grain), picrotoxin ($\frac{1}{80}$ to $\frac{1}{40}$ grain), aromatic sulphuric acid (5 to 10 drops), and camphoric acid (5 to 10 grains).

℞. Atropinæ sulphatis . . . . gr. ⅛
Acidi sulphurici aromatici . . . . . . fʒiij
Aquæ menthæ piperitæ . . . . . q. s. f℥iij.—M.
SIG.—Teaspoonful in water at bedtime.

Pyrexia.—In many cases the fever yields to absolute rest in bed or in a reclining chair, combined with life in the open air. Cold sponging is useful when the temperature is high. In obstinate cases the administration of phenacetin (3 to 5 grains) may be tried.

Pleuritic Pains.—Mild attacks generally yield to sinapisms or the application of iodin. Strapping the affected side also affords relief. Severe pains should be treated by the application of small blisters and the subcutaneous administration of morphin.

Diarrhea.—Diarrhea, the result of indigestion, usually yields promptly to restriction of the diet, rest, and the administration of a mild mercurial. Persistent diarrhea will demand the use of bismuth subnitrate (20 to 30 grains) combined with opium and intestinal antiseptics—salol, bismuth-beta-naphthol, or creasote. Combinations of tannigen (5 to 15 grains) or tannalbin (5 to 15 grains) with bismuth compounds are also useful :

℞. Tannigen . . . . ʒj
Bismuth-beta-naphthol . . . . ʒij
Codeinæ sulphatis . . . . gr. v.—M.
Fiant chartulæ No. xij.
SIG.—One every four hours.

Hemoptysis (see p. 240).

DISEASES OF THE PLEURA.

PLEURISY.

(**Pleuritis.**)

Definition.—Inflammation of the pleura.

Varieties.—According to cause, it may be divided into primary or secondary; according to extent, into unilateral, bilateral, or local; according to time, into acute or chronic; and according to the exudation, into serofibrinous, fibrinous, or purulent.

Etiology.—Pleurisy may be: (1) Idiopathic, arising from exposure to cold and wet. (2) Traumatic. (3) Secondary to inflammatory diseases of adjacent viscera, as pneumonia and phthisis. (4) Secondary to some general morbid process, as rheumatism, Bright's disease, and the infectious fevers. (5) Tuberculosis. (6) Cancerous (rare).

At least three-fourths of all cases of serofibrinous pleurisy are tuberculous.

Pathology.—In the early stage the membrane is red, sticky, lusterless, and covered with a thin film of lymph; if the process now ceases, the condition is termed *dry pleurisy*. If, however, the inflammation continues, an exudate is formed which may be: (1) Serofibrinous; (2) fibrinous; or (3) purulent (empyema). In the *serofibrinous* form there is little lymph, the exudate being mainly composed of straw-colored serum (a few ounces to several pints) which in favorable cases is gradually absorbed. In large effusions the adjacent organs are displaced and the lungs are compressed. In the *fibrinous* form serum is scant and the membrane is covered with a butter-like exudate that subsequently organizes and unites more or less closely the pleural surfaces, causing *chronic pleural thickening*. A liquid effusion, which is circumscribed and confined to pockets formed by adhesions, is termed *sacculated pleurisy*.

Purulent pleurisy is always the result of micro-organismal infection. Left to itself, it may kill by sepsis, it may become inspissated and encysted (very rare), or it may rupture spontaneously into the lung and bronchi, or more rarely through the chest-walls. After the discharge of the pus, the pleural surfaces may eventually become united by firm adhesions.

Hemorrhagic Pleurisy.—A bloody effusion is often observed in tuberculous and cancerous pleurisies and in pleurisy associated with scurvy, grave anemia, and other cachectic states.

Symptoms.—The disease usually sets in with a sharp stabbing *pain* in the side, aggravated by deep breathing. The respirations are rapid and shallow.

Fever is moderate in degree (101° to 103° F.).

There is a slight *irritative cough*, but no expectoration. As the effusion accumulates, the pain diminishes, but dyspnea and cyanosis gradually develop.

In some cases (*latent pleurisy*) the disease begins insidiously, weakness and dyspnea being the first symptoms to attract attention.

Physical Signs.—*Inspection.*—In the first stage there may be deficient expansion on the affected side, owing to the severe pain. After the development of a liquid effusion the characteristic features are immobility, bulging of the intercostal spaces, and displacement of the apex-beat.

Palpation reveals immobility of the affected side and absence of the vocal fremitus.

Percussion yields marked dulness or flatness and a sensation of increased resistance. The upper line of dulness is not horizontal, but is curved and rises higher posteriorly. In moderate effusions the level of dulness often changes with the position of the patient. Above the effusion percussion gives a tympanitic note (Skoda's resonance). In left-sided effusions Traube's semilunar space is obliterated.

Auscultation.—In the early stage this detects a to-and-fro friction-sound of respiratory rhythm. After the development of the effusion the respiratory sounds are weak and distant. Occasionally they have a tubular quality, especially near the margins of the liquid. Vocal resonance is usually diminished or absent, but occasionally, when the effusion is moderate, egophony may be heard. The friction-sound may again be audible when the fluid disappears.

Mensuration shows an increase in the size (½ to 1 inch) of the affected side.

Diagnosis.—**Croupous Pneumonia.**—The severe chill, rusty expectoration, high fever, the fine inspiratory râles, dulness not changing with the patient's posture, increased vocal fremitus, increased vocal resonance, loud bronchial breathing, and the absence of bulging and of displaced apex-beat will serve to distinguish pneumonia from pleurisy.

Pleurodynia (Rheumatism of the Intercostal Muscles).—In this affection the pain and tenderness are diffuse; moreover, fever, friction-sounds, and signs of effusion are absent.

Diaphragmatic Pleurisy.—This may present the following symptoms: Intense pain under the margin of the ribs, with tenderness on pressure; thoracic breathing; tenderness over the phrenic nerve, which is accessible between the two roots of the sternocleidomastoid at the base of the neck; hiccup; and severe dyspnea. The physical signs are not marked.

Pericarditis with Effusion.—In this condition the percussion-dulness has a characteristic shape, the sounds of the heart are distant and muffled, and there is greater embarrassment of the circulation.

Hydrothorax.—In this condition pain and fever are absent. There is often a history of cardiac or renal disease, and the fluid on aspiration is found to contain less than 3 per cent. of albumin and to have a specific gravity below 1015.

Pyothorax.—This may be recognized by the general symptoms of sepsis—persistent irregular fever, increasing pallor, profuse sweats, chills, and leukocytosis. In doubtful cases it will be necessary to aspirate.

Prognosis.—In simple serofibrinous pleurisy the prognosis is guardedly favorable. Fever usually subsides in from a week to ten days, and absorption of the fluid in most cases is complete in from four to six weeks. Sudden death occasionally occurs when the fluid is excessive. In about one-third of the cases tuberculosis sooner or later develops.

Treatment.—The patient should be kept in bed and restricted to a liquid diet. Mercurial or saline aperients may be prescribed at the onset. For the severe pain the application of a blister or of wet or dry cups, together with the administration of morphin, will be found effective. Strapping the affected side with broad strips of adhesive plaster is also useful. Acute sthenic cases with decided fever are often favorably influenced by the administration of salicylates (1 to 1½ drams of the sodium or ammonium salt a day). In asthenic cases salicylates are of no avail.

Removal of Serous Effusion.—The most useful measures for promoting absorption are the application of iodin or of flying blisters, and the administration of hydragogue cathartics and of diuretics. From ½ to 1 ounce of magnesium sulphate may be given in as little water as possible an hour before breakfast, and the fluid consumed by the patient during the day restricted to a minimum. The most serviceable diuretics are digitalis, caffein, and potassium acetate. Potassium iodid (5 to 10 grains thrice daily) is also employed for its absorbent effect. Diaphoretics are of little vlaue.

Paracentesis is demanded—(1) When the effusion is considerable and shows no signs of receding after the lapse of two weeks; (2) when there is sufficient fluid to cause severe dyspnea, cyanosis, persistent cough, or failing pulse; (3) when the fluid reaches the level of the second rib and there is marked dislocation of the neighboring organs; (4) when the presence of pus is suspected.

The most favorable site for the puncture is usually in the sixth or seventh intercostal space, between the mid-axillary line and the angle of the scapula. After anesthetizing the part, the needle should be introduced with a quick stroke along the upper margin of the rib. The fluid should be removed slowly, and under no circumstance should extreme efforts be made to obtain the largest possible amount. The operation should be terminated at once if incessant cough, severe pain, dyspnea, palpitation, tendency to syncope, or other untoward symptoms appear.

Empyema (Pyothorax).—The effusion may be primarily purulent, having been excited by pyogenic microorganisms, or a serofibrinous effusion, through subsequent infection, may become purulent. Traumatism or the rupture of a purulent accumulation into the pleural sac is an occasional cause. It frequently follows pneumonia, particularly in children, in whom the most common form of pleurisy is empyema. It is often secondary to tuberculosis or one of the infectious fevers.

The organisms most frequently present are the pneumococcus, staphylococcus, streptococcus, tubercle bacillus, and typhoid bacillus.

Symptoms.—The physical signs and symptoms are similar to those observed in serofibrinous pleurisy. Pus is indicated by septic phenomena—high and irregular fever, sweats, chills, pallor, and leukocytosis; by the results of aspiration; and sometimes by edema of the chest-walls. In pulsating pleurisy the effusion is almost always purulent.

Prognosis.—Grave, though recovery frequently occurs. The most favorable cases are those following pneumonia.

Treatment.—This consists in free incision and thorough drainage. Irrigation is unnecessary unless the fluid is putrid. In long-standing cases the excision of several ribs (Estlander's operation) facilitates retraction and the obliteration of the pleural sac, which is essential to a cure.

HYDROTHORAX.

Definition.—A serous exudation of non-inflammatory origin in the pleural cavity.

Etiology.—It is always secondary. It may result from one of the causes of general edema—heart disease, nephritis, emphysema, or anemia; it may be due to pressure upon the veins by a tumor, aneurysm, or a dilated right auricle from mitral disease. It is usually bilateral, but when caused by pressure or mitral lesions, it is frequently unilateral.

Symptoms.—It gives rise to dyspnea, cyanosis, and the physical signs of a pleural effusion.

Diagnosis.—This is based upon the history, the absence of pain and fever, and the character of fluid obtained by aspiration (see p. 268).

Treatment.—Remedies should be directed to primary disease. If hydragogue cathartics and diuretics fail to afford relief and the dyspnea becomes urgent, aspiration must be practised.

PNEUMOTHORAX.

Definition.—Air in the pleural cavity.

Etiology.—About 90 per cent. of the cases result from the rupture of a tuberculous cavity into the pleura. Rupture of the lung from abscess, gangrene, or emphysema is a comparatively rare cause. It may occur after the spontaneous rupture into the lung of an empyema. It is occasionally due to penetrating wounds of the chest.

Pathology.—The adjacent viscera are often much displaced and the lung is compressed. Even when air alone has escaped into the pleural sac, an effusion soon forms, so that *pneumohydrothorax* or *pneumopyothorax* is an almost inevitable result.

Symptoms.—The onset is usually marked by sharp pain, severe dyspnea, cyanosis, cough, and the symptoms of shock—subnormal temperature, a weak, rapid pulse, cold extremities, and pinched features.

Physical Signs.—*Inspection* may reveal distention of the affected side, immobility, and marked displacement of the apex-beat.

Palpation.—Vocal fremitus is diminished.

Percussion.—Over the air there is tympany. At the base there may be flatness, changing with the posture of the patient.

Auscultation.—The respiratory murmur and vocal resonance are usually absent, but when the opening in the lung remains patulous, amphoric breathing may be detected. Metallic tinkling is often heard. When a silver coin is placed on the affected side and is struck with another, the auscultator may detect a clear metallic sound (bell-tympany). When fluid is present, shaking the patient elicits a splashing sound (Hippocratic succussion).

Diagnosis.—**A Large Phthisical Cavity.**—This is usually located near the apex instead of the base; the surface is sunken, not prominent; the heart is not displaced; succussion-splash and bell-tympany are rarely obtainable.

Dilated Stomach.—This may give a tympanitic note over the left pulmonary base, and may simulate a pneumothorax; but the tympanitic note is continued down into the abdomen, and the swallowing of liquid is distinctly audible over the base of the chest.

Prognosis.—This depends on the cause. In tuberculous subjects it almost always proves fatal in from a few days to a few months. In empyemic and traumatic cases the outlook is distinctly more favorable.

Treatment.—In tuberculous cases the indications are to relieve distress by morphin and to combat collapse by such stimulants as ether, ammonia, camphor, alcohol, and strychnin. Aspiration occasionally affords temporary relief. In non-tuberculous cases of pneumopyothorax operative interference is generally advisable.

HEMOTHORAX.

Definition.—Blood in the pleural cavity.

Etiology.—It usually results from wounds of the chest-wall, fracture of the ribs, or the rupture of an aneurysm. A sanguineous inflammatory (*hemorrhagic pleurisy*) exudate frequently occurs in cancerous and tuberculous pleurisy and in simple pleurisy when the individual is profoundly anemic.

Symptoms.—The symptoms and physical signs are those of pleural effusion.

ACUTE INFECTIOUS DISEASES.

FEVER.

FEVER is an abnormal condition, characterized by elevated temperature, quickened respiration and circulation, faulty secretions, and increased tissue-waste. It is dependent upon a perversion of the physiologic processes whereby the generation and the loss of heat are so balanced as to maintain a uniform normal temperature.

The Detection of Fever.—There is only one reliable way of detecting fever, and that is by means of the clinical thermometer. The instrument may be placed in the axilla, mouth, rectum, or vagina.

When the axilla is selected, the following precautions must be observed: Wipe off the perspiration and dry the skin; insert the bulb of the instrument deep in the armpit, and see that the arm is kept close to the side. The thermometer should be kept in position until the mercury maintains the same level for two minutes; this will usually require in all about six or seven minutes.

When the mouth is selected, the bulb should be placed under the tongue and the lips kept closed. Hot or cold drinks recently taken mar the result. For obvious reasons the mouth should not be used in delirious patients.

The rectum may be selected in children. The rectal temperature is about a degree higher than that of the axilla.

Febrile Stages.—The course of all fevers is marked by three stages: (1) Invasion; (2) fastigium, or stadium; (3) defervescence, or decline.

Invasion.—During this period the temperature gradually rises until it reaches its maximum.

Fastigium.—In this period, though there may be marked variations, the temperature shows a tendency to touch again and again its highest point.

Defervescence.—In this period the temperature gradually falls until it reaches the norm.

Terminations of Fever.—Fever terminates by lysis or crisis.

Lysis.—The temperature falls slowly by slight gradations until it reaches the norm.

Crisis.—The temperature falls suddenly—often four or five degrees in twelve or twenty-four hours.

The Degree of Pyrexia.—The following is Wunderlich's classification of febrile temperatures:

1. Subfebrile, temperature 99.5°–100.4° F.
2. Slightly febrile, temperature 100.4°–101.3° F.
3. Moderately febrile, temperature 101.3°–103.1° F.
4. Decidedly febrile, temperature 103.1°–104° F.
5. Highly febrile, temperature above 103.1° F. in the morning and above 104.9° F. in the evening.
6. Hyperpyretic, temperature above 106° F.

Febrile Remissions.—All fevers show a diurnal variation. The maximum is usually reached at about 6 P. M. and the minimum at about 6 A. M. Occasionally these extremes are reversed and the maximum is in the morning and the minimum in the evening. The daily difference amounts to about 1° F.

Types of Fever.—According to the degree of the diurnal variation three types are recognized:

1. *Continued Fever.*—The diurnal variation is slight—1°–1.5° F. Typhus fever, pneumonia, and scarlet fever are examples of continued fevers.

2. *Remittent Fever.*—The diurnal variation is marked, but the minimum temperature is still above the norm. Typhoid fever, remittent fever, and septic fever are examples of this type.

3. *Intermittent Fever.*—The diurnal variation is marked, and the minimum is normal or subnormal. The following fevers show multiple intermissions:

1. Intermittent malarial fever.

2. Relapsing fever.
3. Septicemic fever (this may be intermittent or remittent).
4. Hepatic intermittent fever (see p. 100).

A *single intermission or marked remission* is observed in the following fevers:

Smallpox (a remission occurs about the third day).

Yellow fever (an intermission or decided remission occurs about the third or fourth day).

Measles (a distinct remission often occurs on the second or third day).

Dengue (an intermission occurs about the third or fourth day and lasts two or three days).

Causes of Fever.—The chief cause is disturbance of the heat-regulating centers by toxic substances circulating in the blood. These substances may owe their origin to bacterial invasion (acute infectious diseases), to faulty metabolism (acute gout, thermic fever), or to mechanical, thermic, or chemical injury of the tissues. Occasionally, as in hysteria, fever appears to be due to a direct disturbance of the heat-regulating centers.

Symptoms of Fever.—The temperature is elevated, the pulse is accelerated, the respirations are increased, the tongue is coated, the appetite is impaired, the secretions of the alimentary canal are deficient, and the urine is scanty, dark colored, and of high specific gravity. Persistent fever is attended with great wasting of the body.

The pulse-temperature ratio:

A temperature of	98.4° F.	corresponds to a pulse of	70.
" "	100° F.	" " "	80–90.
" "	102° F.	" " "	100–110.
" "	104° F.	" " "	120–130.

Effects of Fever on the Tissues.—High and long-continued fever induces certain marked changes in the tissues, especially cloudy swelling, fatty degeneration, and coagulation necrosis.

Treatment of Fever.—Febrile patients should be kept at rest in a cool, well-ventilated room. The diet should be liquid or semiliquid. When the fever is moderate, no special treatment is required, but sponging with cool water or with alcohol and water and the administration of such drugs as spirit of nitrous ether, solution of ammonium acetate, or neutral mixture afford comfort. High fever is best controlled by cold: cold sponging, the cold pack, or the cold bath.

In applying the *cold pack* the bedding is first protected by water-proof sheeting; the patient is then stripped and enveloped in an ordinary sheet wrung out of water at a temperature of 70°–60° F. The pack is usually continued for from ten to fifteen minutes, and during this time it is necessary to sprinkle the sheet at frequent intervals with water sufficiently cool to maintain a uniform temperature.

The Cold Bath.—The patient is wrapped in a sheet and then placed in water at 70° F. While in the bath an ice-cap is kept upon the head and the trunk and limbs are vigorously rubbed, so as to bring new relays of blood to the surface. A stimulant is sometimes given before the bath to lessen the shock. At the end of fifteen or twenty minutes the patient is carried back to bed and covered with a dry sheet and a light blanket. After he has been thoroughly dried the damp coverings are removed and replaced by dry ones. If the patient be delicate, it is preferable to place him in a bath at 90° F. and then gradually lower the temperature of the water to 70° F.

Drugs may also be used to lower temperature, but the application of cold is generally preferable. Phenacetin, antipyrin, acetanilid, and quinin are the antipyretic drugs most commonly employed.

Period of Incubation.—The period elapsing between the occurrence of the infection and the development of symptoms.

It varies considerably in the same disease, being more or less influenced by the susceptibility of the patient and the virulence of the contagion. The average period of incubation in the various fevers is as follows:

Typhoid fever: two to three weeks.
Typhus fever: a few days to two weeks.
Measles: ten days to two weeks.

Rötheln or rubella: one to three weeks.
Scarlatina: two to seven days.
Smallpox: ten days to two weeks.
Erysipelas: three to seven days.
Diphtheria: two to seven days.
Varicella: fourteen to sixteen days.
Tetanus: a few days to three weeks.
Mumps: two to three weeks.
Yellow fever: from three to four days.
Cholera: two to five days.

The date at which rashes appear in the various acute infections:

Typhoid fever: seventh to the ninth day.
Typhus fever: fourth or fifth day.
Smallpox: third or fourth day.
Measles: third or fourth day.
Scarlatina: first or second day.
Rötheln or rubella: first or second day.
Varicella: first day.

Protection from Future Attacks.—Few diseases confer absolute immunity against future attacks, but the following are fairly protective:

Typhoid fever: relapses are common, but second attacks are infrequent.
Typhus fever: second attacks are very rare.
Measles: second attacks are uncommon.
Rubella: second attacks are rare.
Scarlet fever: second attacks are rare.
Smallpox: second attacks occasionally occur.
Mumps: second attacks are rare.
Varicella: second attacks are uncommon.
Yellow fever: second attacks are rare.

The following specific fevers do not confer immunity:

Erysipelas.
Relapsing fever.
Diphtheria.
Rheumatic fever.
Malarial fever.
Influenza.
Croupous pneumonia.

Termination by Crisis.—The following infectious fevers are apt to end by crisis:

Typhus fever.
Pneumonia.
Malarial fever.
Measles.
Relapsing fever.
Erysipelas.

Infections in which jaundice is likely to occur:

Yellow fever.
Relapsing fever.
Acute yellow atrophy of the liver.
Remittent malarial fever.

SUBNORMAL TEMPERATURE.

Temperatures below 97.5° F. may be considered subnormal. They are observed in the following conditions:

1. During convalescence from certain febrile diseases. After pneumonia and typhoid fever the temperature may remain subnormal for several days.
2. In collapse from various causes.
3. In cholera. In this disease the temperature may be very low (90°–85° F.) for several days.
4. In certain chronic diseases, especially myxedema, diabetes, cancer, chronic cardiac, cerebral, and spinal diseases.

SIMPLE CONTINUED FEVER.

(Febricula; Ephemeral Fever.)

Definition.—An acute febrile disease, of short duration, without definite lesions or a specific etiology.

Etiology.—It is generally met with in young and sensitive individuals. Exposure to the sun, prolonged physical or emotional excitement, and errors in diet seem to excite it.

Symptoms.—The disease usually begins abruptly with chilliness, headache, malaise, and fever which soon attains a maximum of 102° or 103° F. The face is flushed; the pulse is full and rapid; the urine is scanty and high colored; the tongue is coated; the appetite is lost; and the bowels are constipated. There is no characteristic eruption, but herpes is frequently observed on the lips.

The disease lasts from a few days to two weeks, and may end by crisis or lysis.

Diagnosis.—Care must be taken to exclude local inflammations, such as gastritis, tonsillitis, and pneumonia.

Typhoid Fever.—At first the diagnosis may be impossible, but the absence of diarrhea, tympanites, abdominal tenderness, splenic enlargement, Widal reaction, and eruption will soon make the diagnosis apparent.

Remittent Fever.—The history, the splenic enlargement, and the presence of hematozoa in the blood will serve to distinguish this disease from simple continued fever.

Prognosis.—Favorable.

Treatment.—The patient should be confined to bed and placed upon a liquid or semiliquid diet. Fractional doses of calomel may be employed to relieve constipation.

The fever is best controlled by sponging with water and alcohol and by the use of some mild refrigerant mixture like the following:

℞. Tincturæ aconiti . . . . ♏xxiv.
Spiritus ætheris nitrosi . . . . fʒv
Liquoris ammonii acetatis . . q. s. ad f℥iij.—M.
SIG.—A dessertspoonful every two hours for a child of four years.

TYPHOID FEVER.

(**Enteric Fever ; Typhus Abdominalis.**)

Definition.—An acute infectious disease, excited by a special bacillus, characterized anatomically by definite lesions in Peyer's patches, mesenteric glands, and spleen ; and manifested clinically by fever, headache, delirium, abdominal distention and tenderness, diarrhea, enlargement of the spleen, and a rose-colored rash.

Etiology.—Early adult life (second and third decades), individual susceptibility, either acquired or hereditary, and bodily fatigue are predisposing factors. The disease is most prevalent during the late summer and early fall.

The exciting cause is the *Bacillus typhosus.* To produce enteric fever it must gain access through the alimentary canal. The fecal discharges and the urine of the patient are the source of the contagion, and drinking-water contaminated by them is the chief medium of transmission. Milk contaminated after leaving the cow is a fruitful source of infection. Excremental contamination may cause infection also through the medium of certain articles of food, such as oysters, celery, and lettuce. Flies may be an important agent in disseminating the disease. Occasionally nurses, physicians, and washerwomen are infected directly.

Pathology.—The characteristic lesions are found in the abdominal lymphatics, namely, in Peyer's patches, solitary glands, and mesenteric glands. The changes in Peyer's glands are best studied in the lower part of the ileum, which should be opened on the side of the mesenteric attachment.

In the first few days the glands are swollen and hyperemic; later there is a marked cell-proliferation, the blood-vessels are compressed, and the glands become pale and prominent (medullary infiltration). If the disease advances, necrosis sets in, the glands becoming yellow and soft. In a few days the necrotic tissue is discharged, leaving an oval, ulcerated surface with somewhat irregular margins, and a smooth base formed by the submucous coat, muscular coat, or peritoneum.

In the fourth week cicatrization begins, and the gland is ultimately replaced by a smooth depressed scar.

In addition to these glandular lesions the mucous membrane of both large and small intestines shows catarrhal changes.

In mild cases the stage of ulceration may not be reached, the proliferated cells being removed by fatty degeneration and absorption without rupture of the gland. The solitary and mesenteric glands pass through similar changes, but the latter rarely rupture. Other lesions are found that are not characteristic. The spleen is soft and swollen. The liver, kidneys, and heart reveal parenchymatous degeneration. The respiratory tract is commonly the seat of catarrhal inflammation.

In rare instances there appears to be a general infection without lesions of the intestinal glands (*typhoid septicemia*).

Period of Incubation.—Two to three weeks.

Symptoms.—*Prodromal Symptoms.*—These consist in gradual weakness, headache, vague pains, nose-bleed, and often slight diarrhea.

The Attack.—*Fever.*—The temperature rises gradually, reaching its maximum (104°–105° F.) by the end of the first week; it remains at this elevation for another period of from one to two weeks, when a gradual defervescence begins and occupies a third period lasting from one to two weeks. Throughout its course the fever is characterized by marked daily remissions, the evening temperature being from one to three degrees higher than the morning.

In some cases, especially in the young, the temperature rises quite abruptly. Slight diurnal remissions indicate a protracted case. As defervescence advances the temperature becomes more irregular; the remissions are more decided, and not infrequently the higher temperature is recorded in the morning. An abrupt fall of several degrees should suggest intestinal hemorrhage or perforation.

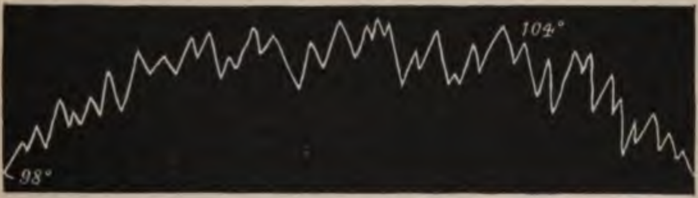

FIG. 13.—Temperature-curve in typhoid fever.

Respiratory Symptoms.—These include hurried breathing, slight cough, and bronchial râles.

Circulatory Symptoms.—The pulse becomes rapid, weak, and dicrotic. The rapidity is often less than such temperatures generally produce. The heart-sounds become feeble. The first may be especially weak, and resembles the second.

The Face.—The expression is dull and heavy, the cheeks are somewhat flushed; the conjunctivæ are clear, and the pupils dilated.

The tongue is tremulous; at first it is red at the tip and edges, and covered posteriorly with a whitish fur. In severe cases the tongue becomes dry, brown, and fissured, and *sordes* collect on the teeth.

The Stomach.—Gastric symptoms are not common, but obstinate vomiting sometimes develops and becomes a serious complication.

Intestinal Symptoms.—The abdomen is distended. Tenderness is frequently noted on palpation; it may be general or confined to the right iliac fossa. Gurgling may also be detected in the latter region, but it has little significance. Diarrhea is generally present, though it is not a constant symptom. The discharges vary in number from three to six or more a day; they are thin, offensive, and of a yellowish color (likened to pea-soup); on standing, a turbid liquid rises to the top and a granular sediment falls to the bottom.

The Eruption.—This appears from the seventh to the ninth day, and is most abundant on the abdomen, though it is not infrequently observed on the chest and back. It is composed of small, slightly elevated, rose-colored spots that disappear on pressure. It comes out in successive crops over several days. It may be absent particularly in the old and very young. Rarely, in malignant cases, is the eruption petechial.

Sudamina are also noted, and result from free perspiration.

Splenic enlargement is rarely absent. Rupture has occurred in a few instances.

Nervous Symptoms.—In mild cases apathy, headache, and slight deafness may be the only nervous symptoms. In severe cases there may be muttering delirium, stupor, twitching of the tendons (subsultus tendinum), picking at the bedclothes or imaginary objects (carphologia), and coma.

The Blood.—The red cells and hemoglobin are reduced. There is no leukocytosis, but, on the contrary, leukopenia.

Widal Reaction.—Blood-serum of typhoid patients when mixed with a fresh bouillon-culture of the typhoid bacillus, after the lapse of a few hours, clears the liquid and throws down a flocculent precipitate. Microscopic examination shows that this precipitation is due to a loss of the motility of the bacilli and their agglutination or aggregation in clumps.

The reaction does not appear, as a rule, before the end of the seventh or eighth day, and may persist for several months or years after recovery. It can be obtained from dried blood or from blood collected in a glass tube of small caliber. As a means of diagnosis it is reliable only when the serum is mixed with the bouillon-culture in no greater proportion than 1 to 40. Of 2283 typhoid cases, 95.57 per cent. yielded the reaction; of 1365 non-typhoid cases, there was no reaction in 98.4 per cent.

The urine is febrile and often slightly albuminous. In many cases (20 per cent.) it contains typhoid bacilli. Retention is common.

Convalescence is marked by anemia, falling of the hair, desquamation of the cuticle, and sometimes by mental enfeeblement.

Varieties.—Mild Typhoid.—There is moderate fever with marked remissions; the diarrhea is slight; nervous symptoms are often absent; the rash is usually present, and may be abundant.

Abortive Typhoid.—There is an abrupt onset with severe symptoms, but convalescence follows in from ten days to two weeks.

Walking Typhoid.—The symptoms are mild, and often disregarded by the patient, who refuses to go to bed; but grave symptoms may develop suddenly, and death from perforation is not uncommon.

Typhoid in Children.—The rash is often absent; the fever rises abruptly; cerebral symptoms are frequently marked.

Complications.—Any symptom aggravated constitutes a complication; thus high fever, excessive diarrhea, and tympanites may be troublesome complications.

Hemorrhage.—This usually occurs during the third week, and is indicated by a sudden fall of temperature, followed by dark-red or tarry stools.

Perforation.—This occurs in about 2 per cent. of all cases. It may be recognized by sudden localized pain and tenderness, fall of temperature, leukocytosis, marked tympanites, disappearance of the liver dulness, vesical irritation, and signs of peritonitis.

Pneumonia (croupous or catarrhal) and *hypostatic congestion of the lungs* are common complications.

Among the less frequent complications or sequelæ may be mentioned: Neuritis, nephritis, pyelitis, cholecystitis, appendicitis, otitis media, periostitis, parotitis, phlebitis, and temporary insanity.

Relapse and Recrudescence.—*Relapses* are quite common; they repeat the symptoms of the original attack, but they are generally milder and of shorter duration, and seldom prove fatal.

Recrudescence.—This is a sudden temporary elevation of temperature occurring during convalescence, and is not associated with a return of the other symptoms. It is usually due to constipation, excitement, or irritating food.

Diagnosis.—**Acute miliary tuberculosis** often closely resembles typhoid fever. In tuberculosis the temperature is generally more irregular; the abdominal symptoms are less marked; pulmonary symptoms, especially dyspnea, are more marked; the rash is absent; the Widal reaction is absent; tubercles may be detected on the retina; and symptoms of basilar meningitis may be present, such as irregular pupils, ptosis, and strabismus.

Ulcerative Endocarditis.—The diagnosis may be impossible, but the following features would suggest endocarditis: The history of a primary disease which might induce ulcerative endocarditis; irregular fever; intercurrent rigors; marked leukocytosis; precordial pain and endocardial murmurs; and the absence of a rose-colored rash, of the Widal reaction, and of marked abdominal symptoms.

Enteritis.—The absence of high fever, of eruption, of splenic enlargement, of epistaxis, and of bronchial catarrh will serve to distinguish enteritis from typhoid fever.

Meningitis.—The abrupt onset, the early development of cerebral symptoms, the irregular fever, the leukocytosis, and the absence of the characteristic rash, of abdominal symptoms, and the Widal reaction will indicate meningitis.

Prognosis.—The prognosis should always be guarded. No case is too mild to prove fatal, and no case is too severe to recover. The mortality varies in different epidemics.

Under present methods of treatment the average mortality is about 8 per cent.

Continued high fever with slight diurnal remissions, excessive diarrhea, severe cerebral symptoms, and repeated hemorrhages are unfavorable features.

Treatment.—As soon as the nature of the disease is recognized the patient should be confined to bed. The room should be large and airy, and provided with efficient means of securing thorough ventilation. The temperature of the room should be maintained between 65° and 70° F. The bed-pan must be used from the beginning until convalescence is well advanced. The stools and urine should be rendered innocuous before being disposed of. This may be done by treating the evacuation with twice its volume of a 1 per cent. solution of chlorinated lime or a 5 per cent. solution of carbolic acid, and allowing it to stand in a covered vessel for two hours before emptying it into the closet. Soiled clothing should be thoroughly boiled.

The diet should be liquid or semisolid, unirritating, and easily assimilable. As a rule, milk is the best food. Most patients will be able to take from 2 to 4 pints in the twenty-four hours, given in portions of from 4 to 6 ounces every two or three hours. It is generally advisable to dilute the milk with lime-water. If curds appear in the stools, the quantity of milk should be reduced. Among other permissible articles may be mentioned buttermilk, kumiss, junket, milk-whey, ice-cream, albumin-water, oyster, mutton, or chicken-broths, chicken jelly, and consommé. The return to solids should not be commenced, as a rule, until the temperature has been normal for a week. Cool water or ice will be required to allay thirst, and even if the latter is absent, it is well to give one or the other at regular intervals. When the first sound of the heart weakens and the pulse becomes soft, stimulants should be administered. It is desirable to give the alcohol with the milk so as to stimulate the stomach to digest the latter, and at the same time to diminish the number of administrations of food and medicine. From 4 to 8 ounces of brandy or whisky may be required in the twenty-four hours, the amount being determined by the general effect.

The use of the cold bath or the cold pack will be found an excellent method of controlling fever and of preventing the development of severe nervous symptoms. It is especially valuable as a stimulant to the nerve-centers, and may be employed whenever the temperature exceeds $102\frac{3}{5}$° F. Hemorrhage and perforation contraindicate its use (see page 276).

Heart-failure.—Cold bathing and the timely use of alcohol do much to guard against heart-failure. When the tendency to cardiac failure is pronounced, strychnin may be given in doses of from $\frac{1}{40}$ to $\frac{1}{20}$ grain every three or four hours. In severe cases the drug should be given hypodermically. Digitalis or strophanthus may also be tried, but in the presence of fever these remedies often prove ineffectual. If collapse is threatened, ether, alcohol, or, better still, camphor (1 to 2 grains in sterile olive oil) may be given subcutaneously every two or three hours.

Diarrhea.—When the diarrhea exceeds three or four stools a day, a suppository of opium ($\frac{1}{2}$ to 1 grain) may be used once or twice daily. If the diarrhea be troublesome, bismuth subnitrate or silver nitrate may be given by the mouth in combination with opium:

℞. Morphinæ sulphatis . . . . gr. j
Orphol . . . . ʒiss
Bismuthi subnitratis . . . . ʒiij.—M.
Fiant chartulæ No. xv.
SIG.—One powder every three or four hours.

℞. Argenti nitratis . . . . gr. iij
Pulveris opii . . . . gr. vj.—M.
Fiant pilulæ xij.
SIG.—One pill every three or four hours.

In very obstinate cases copper sulphate with opium in pill proves efficacious.

Constipation.—This may be relieved by enemas of soap and water or by the administration of fractional doses of calomel.

Tympanites.—This may be relieved by the application of turpentine stupes and the internal administration of some antiseptic, such as salol, creosote, or bismuth-beta-naphthol.

When extreme, a soft rectal tube may be introduced into the bowel.

Hemorrhage.—Absolute rest is imperative. Cold bathing should be suspended. It is advisable to elevate the foot of the bed. An ice-bag may be applied with advantage to the right iliac region, and ice may be given to suck. The best drug is morphin ($\frac{1}{6}$ to $\frac{1}{4}$ grain) hypodermically. Ergot is of doubtful value. In cases of recurrent hemorrhage gelatin may be given subcutaneously, by the mouth, or by the bowel.

Insomnia.—Opium is generally the best hypnotic. In some cases, however, sodium bromid or chloralamid acts better.

Delirium is best managed by hydrotherapy. Low, muttering delirium usually calls for stimulants. An ice-cap is useful. Camphor or musk may be tried: the former is best given hypodermically, the latter, by the bowel. In active or violent delirium no drug is so generally useful as morphin.

Perforation.—Recovery from peritonitis is so exceedingly rare under medicinal treatment that operative interference is called for in all cases that are not obviously moribund. The operation should be done at the earliest possible moment. Keen has collected 83 cases with 16 recoveries.

TYPHUS FEVER.

(Ship Fever; Jail Fever.)

Definition.—An acute contagious disease unassociated with any characteristic lesions, and manifested by great prostration, a petechial rash, marked nervous symptoms, and high fever that defervesces by crisis in from twelve to fourteen days.

Etiology.—It is excited by an unknown poison that is capable of being carried in clothes (fomites). It is rare in America, but not uncommon in England, Ireland, and Russia. Bad food, impure water, overcrowding, and foul air are predisposing factors.

Pathology.—There are no characteristic lesions. As in other fevers, the liver and spleen are swollen, and the tissues reveal parenchymatous and fatty degeneration. The blood is dark and fluid.

Period of Incubation.—From a few days to two weeks.

Symptoms.—Typhus fever begins abruptly with pain in the head, back, and limbs; extreme prostration; and fever that reaches its maximum (104° to 105° F.) in from two to three days. The temperature remains high until the twelfth or fourteenth day, when it falls by crisis.

The pulse is rapid, weak, and often dicrotic. The tongue is at first moist and covered with a whitish fur, but it soon becomes dry and brown.

The face is dusky; the conjunctivæ are injected; the pupils are contracted.

Nervous Symptoms.—In severe cases these are prominent, and consist of headache, stupor, delirium, subsultus tendinum, carphologia, and coma-vigil.

The Eruption.—About the fourth or fifth day rose-colored spots appear over the body; these rapidly become hemorrhagic or petechial, and fail to disappear on pressure. There is a distinct relation between the amount of eruption and the severity of the attack. In addition to this "mulberry rash" there is often a diffuse, dark-red, subcuticular mottling.

Gastro-intestinal Symptoms.—The stomach is retentive and the bowels are constipated.

Urine.—The urine is scanty, high-colored, and often albuminous.

Complications.—These are not very common, but catarrhal pneumonia, localized gangrene, neuritis, nephritis, and abscesses may occur.

Diagnosis.—**Cerebrospinal Meningitis.**—In this affection the pain in the back is greater; the fever is much more irregular; there is greater tendency to opisthotonos and facial palsies; vomiting is much more common, and the eruption, though it may resemble that of typhus, is inconstant and without a special time for appearing.

Typhoid Fever.—The resemblance is in the nervous phenomena only. In typhoid fever the fever rises and falls very gradually; the eruption appears later, remains rose-red, and does not become petechial; the face is not dusky; the eyes are not injected; the blood yields the Widal reaction, and there are marked abdominal symptoms.

Prognosis.—The mortality varies in different epidemics. It may exceed 20 per cent. Advanced years and alcoholism render the prognosis very grave.

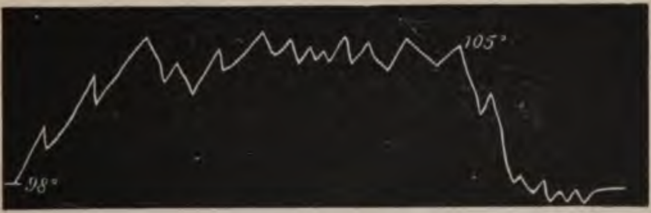

FIG. 14.—Temperature-chart of typhus.

Treatment.—The patient should be isolated, and all the excreta disinfected. The general treatment is the same as that of typhoid fever.

RELAPSING FEVER.

(Spirillum Fever; Famine Fever.)

Definition.—An acute contagious disease excited by the Spirochæta Obermeieri, and characterized by recurring paroxysms of high fever lasting for from five to seven days.

Etiology.—The exciting cause is the Spirochæta Obermeieri, a spiral-shaped microbe three or four times as long as the diameter of a red blood-corpuscle. Bad water, poor food, overcrowding, and foul air predispose to epidemics. The disease is highly contagious.

Pathology.—There are no characteristic lesions. The liver and spleen are enlarged, and the latter is frequently the seat of infarctions. There is usually catarrhal inflammation of the stomach and bile-ducts. The spirochæta is found in the blood during life, but only during the paroxysms; after death it is found in all the organs.

Period of Incubation.—From two days to two weeks.

Symptoms.—The disease begins abruptly with a chill, followed by fever, which reaches its maximum (105°–106° F.) in twenty-four hours, and remains high for from five to seven days, when it falls by crisis. After an intermission of five or six days it again rises rapidly and remains high for a similar period. Convalescence usually begins at the end of the second paroxysm, but it may not begin until after the third or fourth. Other noteworthy symptoms are intense pains in the head, back, and joints, and the presence of the spirochæta in the blood. Gastric irritability and jaundice are also common. Not infrequently there is an ecchymotic rash.

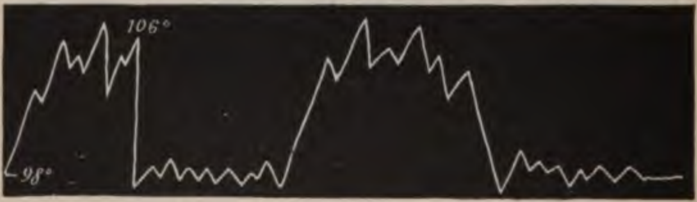

FIG. 15.—Temperature curve in relapsing fever.

Complications.—The chief complications are hyperpyrexia, nephritis, pneumonia, ophthalmia, and hemorrhage from the kidneys, stomach, or bowels.

Diagnosis.—The characteristic febrile paroxysms with the long intermissions and the presence of the spirochæta in the blood are the distinctive features.

Prognosis.—Favorable in uncomplicated cases.

Treatment.—As in all contagious diseases, isolation, free ventilation, and disinfection of excreta and clothing are important safeguards against the spread of the virus. The treatment is purely symptomatic. Absolute rest, good nursing, and proper diet will do much to avert complications. Vomiting may be controlled by carbonated water, wine of ipecac (1 minim), diluted hydrocyanic acid (1 or 2 drops), or cocain ($\frac{1}{8}$ grain); the fever by cold sponging; and the severe pains by morphin or phenacetin.

CEREBROSPINAL FEVER.

(Epidemic Cerebrospinal Meningitis; Spotted Fever.)

Definition.—A specific infectious disease characterized anatomically by inflammation of the cerebrospinal meninges, and clinically by intense pain in the head, back, and limbs, convulsions, irregular fever, and frequently by a petechial eruption.

Etiology.—The disease may be sporadic or epidemic. Overcrowding, poor food, foul air, and bad drinking-water predispose to epidemics. Outbreaks are most common in the winter and spring. The young are more susceptible than the old. The disease is not commonly regarded as transmissible, but cases have recently been reported which furnish strong evidence of its contagiousness.

The Exciting Cause.—The Diplococcus intracellularis of Weichselbaum is the specific cause of the disease. It appears in the polymorphonuclear leukocytes of the exudate, and may be gotten from the nasal secretion and the fluid obtained by spinal puncture.

Pathology.—In most cases the membranes of the brain and cord are deeply congested and opaque. Lymph and pus are found both at the base and on the convexity of the brain, especially in the fissures and along the blood-vessels. The spinal meninges present similar changes, the posterior surface of the cord being particularly involved.

The liver and spleen are engorged, and the muscles reveal granular degeneration. In rapidly fatal cases the lesions may be very slight.

Symptoms.—**Common Form.**—The disease generally begins abruptly with a chill, followed by vomiting and excruciating pain in the head, back, and limbs. The muscles of the neck and back become rigid and contracted, so that the head is bent backward and the back is straightened; in severe cases the body may be arched in a state of opisthotonos. Kernig's sign is an almost constant phenomenon.[1]

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[1] This consists in an inability to straighten the leg completely when the patient is in the recumbent posture and the thigh is flexed at a right angle with the pelvis.

Delirium is rarely absent, and in severe cases it is followed by stupor and coma.

Involvement of the Cranial Nerves.—Pressure of the exudate upon the cranial nerves may produce the following symptoms: Nystagmus (tremor of the eyeball); strabismus; ptosis; irregular, sluggish pupils; and partial deafness or blindness.

Involvement of the Spinal Nerves.—There is extreme cutaneous hyperesthesia, so that the slightest touch excites pain. The muscles of the extremities are stiff and may twitch, but are rarely palsied. The patellar reflex is usually diminished. The joints are occasionally red, swollen, and painful.

Febrile Symptoms.—The temperature is irregular in its course and indefinite in its duration; ordinarily it ranges between 101° and 103° F., but in some cases it is almost normal, and in others it is very high. The pulse is rapid and full; the bowels are constipated; and the urine may contain albumin and sugar. There is usually rapid emaciation. Polyuria is an occasional symptom.

The Eruption.—The eruption is neither constant nor peculiar. In many cases a blotchy purpuric rash appears over the entire body. Herpes facialis is also frequently observed. In other cases urticaria or a roseolar or erythematous rash appears.

The Blood.—Leukocytosis is always present.

Lumbar Puncture.—In a large proportion of the cases diplococci are found either on microscopic examination or by culture.

The duration is from a few hours to several weeks. In favorable cases recovery is always slow.

Fulminant Form.—There is an abrupt onset, with a chill, followed by vomiting, headache, moderate fever, convulsions, a petechial or purpuric rash, and death in a few hours from collapse.

Abortive Form.—The disease begins abruptly with grave symptoms, but terminates in a few days in recovery.

Intermittent Form.—The fever is characterized by intermissions or marked remissions that occur daily or every other day.

Diagnosis.—**Typhoid Fever.**—The gradual onset, the regular fever, the diarrhea and tympanites, the Widal reaction, and the absence of rigidity, of intense pain in the back and limbs, of facial palsies, of leukocytosis, of Kernig's sign, and of herpes, will serve to distinguish typhoid from cerebrospinal fever.

Typhus Fever.—The regular fever, the absence of intense pain in the back and limbs, of facial palsies, of Kernig's sign, and of muscular rigidity will distinguish typhus from cerebrospinal fever.

Acute articular rheumatism may resemble cerebrospinal meningitis, but the early involvement of the joints, the acid sweats, and the absence of rigidity, of eruption, and of facial palsies, will distinguish it from cerebrospinal meningitis.

Tuberculous Meningitis.—In this disease the onset is less abrupt; there is less tendency to opisthotonos; herpes is rare; petechiæ are always absent. Lumbar puncture affords a reliable means of diagnosis and a primary focus of tuberculosis can generally be detected elsewhere in the body.

Prognosis.—The mortality varies in different epidemics from 20 to 80 per cent. The prognosis should always be guarded; the mildest cases may prove fatal. Severe cerebral symptoms usually indicate a fatal termination.

Complications and Sequelæ.—These include defective vision from inflammation of the cornea or retina or from atrophy of the optic nerve; defective hearing from inflammation of the auditory nerve or from suppurative inflammation of the internal or middle ear; pneumonia; arthritis; aphasia; peripheral palsies; imbecility; chronic hydrocephalus; and persistent headache from chronic meningitis.

Treatment.—Cerebrospinal fever is probably not contagious, hence rigid isolation is not usually regarded as absolutely necessary. It is advisable, however, to disinfect the discharges, bed-linen, etc. The sick-room should be quiet, darkened, and well ventilated. The diet should be liquid and supporting. In some cases, in order to secure the ingestion of enough nourishment, it may be necessary to resort to nutrient enemas or forced feeding by means of a stomach-tube. Cardiac failure must be combated by stimulants, of which the best are whisky and brandy.

In sthenic cases the withdrawal of several ounces of blood by wet-cups applied along the cervical vertebræ may prove useful. Cold applied to the head and along the spine affords considerable relief. Blisters to the nape of the neck are of doubtful value, at least during the irritative stage. Morphin hypodermically is the best drug for the relief of pain, restlessness, spasms, and insomnia. In mild cases bromids may suffice.

Fever is controlled best by cold sponging or the cold pack, or, if the temperature is very high, by systematic cold bathing. Repeated lumbar punctures have been found useful in relieving excruciating headache, delirium, somnolence, and coma.

Tonics—iron, strychnin, and cod-liver oil—are generally indicated during convalescence. Local palsies will require massage and electricity.

MALARIAL FEVER.

(Chills and Fever; Fever and Ague; Paludism.)

Definition.—An infectious disease, excited by a protozoan parasite—the hematozoön or Plasmodium malariæ of Laveran, and characterized by splenic enlargement, fever with periodic intermissions or remissions, chills, and anemia.

Etiology.—Man becomes infected with the organism of malaria usually, if not invariably, through the bite of certain mosquitos, namely, those belonging to the genus Anopheles, which serve as hosts for the parasite. The usual source from which the mosquito derives the parasite is man. The conditions predisposing to infection are those which are favorable to mosquito life, namely, high temperature, humidity, and collections of water undisturbed by winds or currents.

Persons living in high-lying localities are less liable to infection than those living in low lands, because the mosquito does not rise high above the ground. Malaria is more apt to be contracted at night than during the day, because most species of anopheles are nocturnal in their habits. Males being more exposed to infection are more often attacked than females.

Pathology.—Three forms of malarial parasites have been distinguished: (1) Tertian; (2) quartan; (3) estivo-autumnal.

Tertian Parasite.—Several hours after a chill small hyaline bodies with ameboid movements appear in some of the red corpuscles. Later pigment-granules in considerable quantity collect around the periphery of the parasite. During this time the organism increases in size and the corpuscle grows paler. Eventually the parasite may fill almost entirely the red cell. After a time the pigment collects in the center, the ameboid movements cease entirely, and segmentation of the parasite follows, with the formation of a roset-like figure composed of from 15 to 26 parts or spores. The red cell next ruptures, and the spores escape in the plasma, to enter other red cells and go through the same cycle of development. The paroxysms or chills occur at the time of sporulation, and are doubtless due to the production of a toxin. The tertian parasite requires forty-eight hours to complete its cycle of development; hence when a single group of these parasites is present, paroxysms occur every other day (*tertian fever*). If, however, two groups coexist and sporulate on alternate days, a paroxysm occurs daily (*quotidian fever*).

Certain mature parasites (sexual forms or gametocytes) do not undergo segmentation, but complete the cycle of their development in the tissues of another host (mosquito). Spores equivalent to those resulting from the segmentation of the asexual forms are inoculated into man by the bite of the insect and on entering the red blood-cells develop into sexual or asexual forms.

Quartan Parasite.—This is less widely distributed than the tertian parasite. It differs from the latter in having coarser but less abundant pigment, and in not decolorizing the red cell. Segmentation occurs in about seventy-two hours, with the formation of from 6 to 14 spores. One group of quartan parasites excites a chill every fourth day (*quartan fever*). When two groups coexist, a chill occurs on each of two successive days, separated by one day of intermission (*double quartan fever*). When three groups coexist, a chill occurs every day (*quotidian fever*).

The life-history of the quartan parasite is similar to that of the tertian parasite.

Estivo-autumnal Parasite.—The latter half of the life-cycle of this parasite is carried out in the internal organs. The endocorpuscular form is smaller than that of either the tertian or the quartan, and contains much less pigment. As the parasite develops the corpuscle acquires a peculiar shriveled and "brassy" appearance. Segmentation does not occur in the peripheral blood, but in the spleen and other internal parts. After the infection has lasted a week or more, crescent, ovoid, and round bodies, with central clumps of coarse pigment granules, appear in the corpuscles, which by this time are scarcely more than thin, colorless shells. The crescent and ovoid forms are gametocytes, the later evolution of which occurs in the tissues of the mosquito. The estivo-autumnal parasite varies in the length of time necessary for completing its developmental cycle.

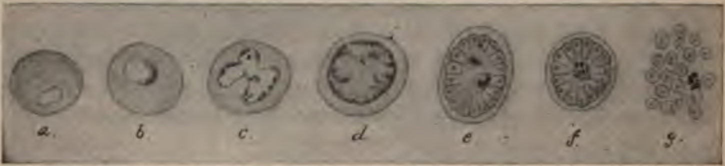

FIG. 16.—Various forms of hematozoa: Tertian organisms (Thayer and Hewetson); *a*, young hyaline form; *b*, hyaline form with beginning pigmentation; *c*, pigmented form; *d*, full-grown pigmented form; *e*, *f*, *g*, segmenting forms.

Pathologic Effects.—The destruction of the red cells by the parasites is followed by anemia, melanemia, and pigmentation of the organs. The spleen becomes greatly enlarged from congestion. In chronic cases (malarial cachexia) it becomes hard and tough from hyperplasia of the fibrous tissue. Extreme disintegration of the blood may occasion thrombosis of small vessels and also hemoglobinuria.

Clinical Varieties of Malaria.—The following clinical forms are recognized: (1) Intermittent malarial fever; (2) estivo-autumnal fever; (3) pernicious malarial fever; (4) chronic malarial cachexia.

INTERMITTENT MALARIAL FEVER.

Intermittent malarial fever is excited by tertian or quartan parasites. It is characterized by paroxysms of fever occurring at definite periods, each paroxysm consisting of a cold, a hot, and a sweating stage.

Cold Stage.—This stage is characterized by lassitude, aching in the limbs, and great chilliness. The features are pinched; the lips are blue; and the surface is cold and rough (cutis anserina). The rectal temperature, however, is high (105°–106° F.). Vomiting may occur. The chill may last from a few minutes to an hour or more.

Hot Stage.—The surface temperature gradually rises; the skin becomes hot; the face flushed; the eyes injected; and the pulse full and rapid. The temperature in the axilla may reach 106° or 107° F. The patient complains of severe pain in the head, back, and limbs, and of intense thirst. The urine is scanty and dark colored. This stage usually lasts from one to five hours.

Sweating Stage.—The fever gradually subsides; the pains grow less, free perspiration follows, and the urine becomes plentiful. Within an hour or two the attack is over and the patient falls into a refreshing sleep.

In addition to the recurring paroxysms, intermittent malarial fever presents symptoms common to all forms of malarial infection, namely, enlargement of the spleen, anemia, pigmentation of the leukocytes, and the presence of hematozoa in the blood. There is no leukocytosis.

ESTIVO-AUTUMNAL FEVER.

(Remittent Fever; Semitertian Fever.)

In temperate zones this type occurs chiefly in the late summer and autumn. In tropical countries, where it often assumes a most severe form, it occurs at all seasons.

The symptoms of estivo-autumnal fever are often quite irregular. The hot stage of the paroxysm often lasts twenty-four or thirty-six hours, or even longer, and the intermissions are very short. In many cases there are no actual intermissions, but simply remissions (remittent fever).

The chill and the sweat may be as severe as in intermittent fever, but usually they are slight and of short duration. There is often slight jaundice (bilious remittent fever). In some cases there is mild delirium, making the condition resemble very closely typhoid fever. Prostration is always marked. The spleen is enlarged. The characteristic parasite is found in the blood.

PERNICIOUS MALARIAL FEVER.

This type is excited by the estivo-autumnal parasite. It prevails in tropical and subtropical countries, and is rare in temperate regions. The second or third paroxysm may assume a pernicious type, but never the first. The symptoms vary with the localization of the parasite. When the latter accumulate in the capillaries of the brain and meninges, the attack may be manifest by delirium, aphasia, and rapidly developing coma (*comatose type*). When the localization is gastro-intestinal, there may be vomiting and purging of serous material, cramps, suppression of urine, coldness of the surface, profuse sweating, and fatal collapse (*algid type*). In other cases, in consequence of a sudden and intense hemolysis, the paroxysms are associated with jaundice, bilious vomiting, and hemoglobinuria. Bleeding into the subcutaneous tissues and from the mucous membranes may also occur (*hemorrhagic type*).

Chronic Malarial Cachexia.—Malarial cachexia may be a sequel of repeated attacks of intermittent or estivo-autumnal fever, or it may develop insidiously as a primary condition.

There is intense anemia with its attending phenomena. Pigment granules are found in some of the leukocytes and in the plasma. The parasites are at times absent from the blood. The complexion is sallow or muddy. The temperature is usually subnormal, but there may be occasional slight attacks of fever. The spleen is greatly enlarged. Weakness and emaciation are marked. Indigestion, flatulency, and constipation are common symptoms. Periodic headache, neuralgia, and hematuria are sometimes observed.

Diagnosis of Malarial Infection.—Estivo-autumnal fever very closely resembles **typhoid fever.** The latter may be recognized by the marked abdominal symptoms, typical rash, Widal reaction, and the absence from the blood of hematozoa.

Yellow Fever.—The comparatively slow pulse, early albuminuria, bloody vomit, single remission in the temperature, and the absence of splenic enlargement and of hematozoa from the blood will serve to distinguish yellow fever from bilious remittent fever.

Charcot's Hepatic Fever.—The history of the case, the pain and tenderness over the region of the gall-bladder, the leukocytosis, and the absence of hematozoa from the blood will lead to the recognition of this condition.

Leukemia.—The marked leukocytosis and the presence in the blood of myelocytes in large numbers will serve to separate myelogenous leukemia from chronic malarial cachexia.

Prognosis.—In the *simple intermittent forms* of malarial fever the prognosis is uniformly favorable. Recovery usually follows in from two to three weeks. *Estivo-autumnal fever* is more resistant, but the outlook is favorable. Pernicious malarial fever is an exceedingly grave disease, the second or third paroxysm not infrequently ending fatally. Many patients with *chronic malarial cachexia* recover under appropriate treatment, but when the spleen is extremely large and the anemia is very pronounced, the prognosis must be guarded.

Treatment.—Prophylactic measures include the extermination of mosquitos, the prevention of infection of mosquitos, and the prevention of infection by mosquitos (Manson). The most useful methods of suppressing mosquitos are the efficient drainage of pools and swamps and the cultivation of damp soils. Covering the surface of the water with petroleum will also free pools from larvas for from two to four weeks. To prevent the infection of mosquitos, malarial patients should be carefully screened. The chief means of preventing infection by mosquitos are avoidance of sleeping in the open air and of exposure to the evening and early morning air, adequate protection from the insects, and the use of quinin in daily doses of from 2 to 5 grains.

Quinin is the only reliable remedy for malarial fever. Methylene-blue (2 to 5 grains with half its weight of powdered nutmeg thrice daily) and Warburg's tincture possess some value, but, being distinctly less efficacious than quinin, they should be employed only when the latter is not well borne.

In ordinary *intermittent fever* the quinin should be given in daily doses of from 15 to 20 grains, so divided that the last dose is taken about three hours before the expected chill.

The remedy should be continued in full doses until the paroxysms fail to appear, and then gradually withdrawn over a period of several weeks. The administration of a laxative dose of calomel as a preliminary measure increases the efficacy of the quinin, probably by facilitating its absorption.

For adults quinin is best prescribed in capsules, cachets, or freshly made pills. For children it may be given suspended in syrup of yerba santa, syrup of chocolate, or elixir of licorice. During convalescence iron and arsenic may be advantageously given with the quinin, as in the following formula:

℞.	Ferri pyrophosphatis	gr. xxx
	Arseni trioxidi	gr. ss
	Quininæ sulphatis	gr. xl
	Pulveris capsici	gr. x.—M.

Pone in capsulas, No. xx.
Sig.—One thrice daily after meals.

In *estivo-autumnal fever* larger doses of quinin (30 to 40 grains a day) are usually required. In *pernicious malarial fever* the patient should be cinchonized as quickly as possible by injecting at once into the tissues of the thigh or buttock about 30–40 grains of a soluble salt of quinin like the dihydrochlorate.

Symptomatic Treatment.—During the cold stage of the paroxysms the patient should be well covered with warm blankets and given hot drinks. Opium in the form of paregoric is sometimes useful in mitigating discomfort. It may be combined with a few minims of aromatic spirits of ammonia, chloroform, or Hoffmann's anodyne. In the hot stage much relief is afforded by frequently sponging the body with cool water, giving cold drinks, and administering, if the symptoms are very severe, a small dose of phenacetin. In the algid type of pernicious malarial fever it may be necessary to give alcohol freely, with digitalis and strychnin, to tide the patient over the paroxysm.

Malarial cachexia requires tonic and hygienic treatment. Arsenic, iron, and cod-liver oil are especially valuable. As in other manifestations of malaria, quinin is indicated so long as the blood shows parasites. According to Wood, it is much better to produce distinct cinchonism at intervals than to give the drug continuously in moderate doses. When there is constipation, mild bitter laxatives are beneficial. Change of locality is sometimes necessary to effect a cure.

SCARLET FEVER.

(Scarlatina.)

Definition.—An acute, contagious disease, characterized by high fever, a rapid pulse, a punctiform scarlet rash, sore throat, and a marked tendency to nephritis.

Etiology.—The specific micro-organism of scarlet fever has not been isolated. The Streptococcus pyogenes is present in most of the complicating lesions. The contagium is usually carried through clothes or other fomites, or in food, particularly milk. The poison is tenacious and of extreme vitality; infected clothes, unused for years, have led to outbreaks. The disease is probably contagious at all periods, but it is most so during the stage of desquamation. The young are especially predisposed, but not equally so. One attack does not give absolute immunity, but second attacks are very uncommon.

Pathology.—The throat is inflamed and sometimes ulcerated; the liver and spleen are engorged; and the muscles reveal granular degeneration. The kidneys frequently show the lesions of hemorrhagic nephritis, the glomeruli being especially involved. The rash is rarely detected after death.

Varieties.—(1) Simple; (2) anginoid; (3) malignant.

Period of Incubation.—From two to seven days.

Symptoms.—The disease generally begins suddenly, occasionally with a chill, but more commonly with vomiting or convulsions.

Throat Symptoms.—These consist in pain and difficulty in swallowing; fullness and tenderness beneath the jaw; and enlargement of the lymphatic glands. The tongue is at first heavily coated and red at the tip and edges; in a few days the coating almost entirely disappears, and the papillæ become bright red and swollen. This appearance has given rise to the term "strawberry tongue." The pillars, tonsils, uvula, and pharyngeal vault are deeply injected and may reveal a punctiform efflorescence before the rash develops on the skin. In severe cases the tonsils may be the seat of follicular inflammation, or may be covered with false membrane.

Eruption.—A scarlet-red punctiform rash appears at the end of the first or at the beginning of the second day, on the neck and chest, and rapidly spreads over the entire body. It disappears on pressure, a white line remaining for a second or two when the finger-nail is drawn through it. It may be uniform or it may occur in discrete patches surrounded by healthy skin. The rash lasts for from five to seven days, and is followed by flaky desquamation. The period of desquamation may last for from two to six weeks.

In some cases the rash is pale and scarcely visible, in others it is slightly papular or vesicular (scarlatina miliaris); in malignant cases it may be petechial.

Febrile Symptoms.—The fever rises abruptly, reaching its maximum (104°–105° F.) in from twenty-four to forty-eight hours, remains nearly uniform for three or four days, and then falls by lysis. The duration of the febrile period is from seven to nine days. The pulse is very rapid—out of proportion to the fever; the respirations are hurried; the appetite is lost; the bowels are constipated; and the urine is scanty, high-colored, and often albuminous. There is a well-marked leukocytosis.

Nervous Symptoms.—Restlessness, headache, insomnia, delirium, and convulsions may occur in the course of the disease. Convulsions developing late in the disease should suggest uremia.

Anginoid Scarlet Fever.—This form is characterized by severe throat symptoms. The tonsils are much swollen and are often covered with false membrane. The fever is high and the prostration is profound. Ulceration of the throat frequently occurs. Death may result from exhaustion, aspiration-pneumonia, or hemorrhage.

Malignant Scarlet Fever.—The onset is abrupt, with a chill, vomiting, or convulsion; the fever is very high (106°–107° F.); the pulse is rapid and feeble; delirium sets in, and is followed by coma. Death may result before the appearance of the rash, in from twenty-four to forty-eight hours.

Complications.—The most common is *nephritis.* This usually develops during convalescence. As mild attacks are unassociated with constitutional symptoms, the urine should be examined daily. Severe attacks are manifested by suppression of urine, general edema, and uremic phenomena. Nephritis may be the immediate cause of death in scarlet fever, or it may become chronic. Many cases, however, end in complete recovery.

Among other complications may be mentioned hyperpyrexia, endocarditis, pericarditis, catarrhal pneumonia, suppuration of the lymphatic glands, ophthalmia, inflammation of the middle ear, and a peculiar inflammation of the joints resembling rheumatism.

Diagnosis.—**Acute tonsillitis** may resemble scarlet fever, especially when the former is associated with an erythematous rash; but in tonsillitis there is no history of contagion, the pulse is proportionate to the fever; the rash, if present, is not punctiform; the tongue has not the strawberry appearance; and there is no tendency to nephritis.

Diphtheria.—The onset is less abrupt; there is more prostration; false membrane, containing the Klebs-Löffler bacillus, is always present; a cutaneous rash is usually absent; and the tongue does not present a strawberry appearance.

Measles.—The sore throat is less marked; catarrhal symptoms are present; the rash appears later, is papular, and forms crescentic-shaped patches; the fever shows a decided remission on the second or third day; and the pulse is proportionate to the fever.

Rotheln.—This may be difficult to distinguish from scarlatina, but the fever is not so high, nor the pulse so rapid; the postcervical glands are more swollen; there is no tendency to nephritis; and the rash is not punctiform.

Accidental Rashes.—Certain drugs like belladonna, quinin, and copaiba, and certain foods, like crabs and oysters, may produce a rash like that of scarlet fever, but it is not punctiform, and is not associated with high fever, sore throat, and rapid pulse.

Prognosis.—Always guarded. The mortality varies in different epidemics from 5 to 40 per cent.

Treatment.—The patient should be isolated for from six to eight weeks. All articles used in the sick-room should be thoroughly disinfected before being removed. To prevent dissemination of the scales some bland ointment (cold cream or cocoa-butter) should be applied to the patient's body at least once a day until desquamation is complete. The patient should not be allowed to leave his bed for at least a week after the fever has subsided.

The diet should consist of milk, junket, kumiss, ice-cream, fruit-juices, and gruels. Water should be given freely to relieve thirst and to keep the secretions active.

Vomiting will call for antiemetics—cracked ice, carbonated water, bismuth subnitrate, or diluted hydrocyanic acid.

Fever.—Tepid sponging is very grateful throughout the febrile period. Fever above 103° F. should be combated with cold packs or baths (80° F.), and by cold applications to the head.

When the temperature is not very high, a mild febrifuge like the following will be found useful:

℞. Spiritus ætheris nitrosi . . . . fʒvj
Liquor ammonii acetatis . . . q. s. ad f℥iij.—M.
SIG.—Dessertspoonful with water every three hours for a child of five years.

Throat Symptoms.—The nose and throat should be cleansed with mild antiseptic sprays, such as a weak Dobell's solution or a solution of hydrogen dioxid (1 : 4).

When tonsillitis is severe, the following application will be found efficacious:

℞. Potassii chloratis . . . . gr. xx
Tincturæ ferri chloridi
Glycerini . . . . aa f℥ss
Aquæ . . . . q. s. ad f℥ij.—M.
SIG.—Apply to the tonsils several times a day with a cotton swab.

Cardiac weakness must be combated with such drugs as alcohol, strychnin, and digitalis.

Cerebral symptoms are best controlled by the application of an ice-cap and the administration of bromids or small doses of chloral or phenacetin. When the nervous symptoms are due to high temperature, cold bathing is most effective.

In *acute otitis media* nothing affords so much relief as gently syringing the auditory canal with hot water. The application of a leech behind the ear is also useful. When the tympanic membrane bulges, indicating the presence of pent-up pus, the latter should be evacuated by puncture.

Should severe *nephritis* develop, dry cupping over the loins, followed by warm fomentations, will often prove of value. Aperients, especially salines, are indicated. Warm baths, hot packs, vapor-baths, or pilocarpin ($\frac{1}{16}$ to $\frac{1}{10}$ grain) should be used to promote diaphoresis. When the urine is scanty, unirritating diuretics, like potassium acetate or bitartrate and digitalis, are of service.

MEASLES.

(Rubeola; Morbilli.)

Definition.—An acute contagious disease, characterized by catarrh of the respiratory tract, moderate fever, and a red papular eruption, which appears on the fourth day, lasts four or five days, and is followed by bran-like desquamation.

Ętiology.—Measles is highly contagious, and the poison may be transmitted through clothes and other fomites. The contagium is apparently associated with the nasal and bronchial secretion, but it has not been isolated. Measles is most commonly observed in children, but unprotected adults are very liable to be attacked. It is essentially an epidemic disease, but now and then sporadic cases occur. One attack usually confers immunity against subsequent attacks.

Pathology.—The lesions consist in catarrh of the entire respiratory tract. Gastro-intestinal catarrh is not uncommon. In fatal cases such complications as catarrhal pneumonia and pulmonary collapse are frequently observed.

Period of Incubation.—Ten days to two weeks.

Symptoms.—The invasion is characterized by *catarrhal symptoms*—photophobia, redness of the eyes, increased lacrimation, sneezing, discharge from the nose, hoarseness, cough, and, in older children, expectoration.

The Fever.—The temperature rises rapidly to 103° or 104° F., but on the second day there is often a decided remission which continues until the fourth day, when the eruption appears; at this time it again rapidly runs up to or beyond its original height, where it remains for three or four days and then falls by rapid lysis or crisis.

The Eruption.—This appears about the third or fourth day on the face, and rapidly spreads over the entire body. It is composed of small, dark-red, velvety papules, which form groups having crescentic borders. There are often much burning and itching of the skin. In three or four days the eruption begins to fade, and a branny desquamation soon follows.

Minute bluish-white specks surrounded by a red areola may be seen on the mucous membrane of the cheeks and lips one or two days before the skin eruption appears (Koplik's sign).

Malignant or Hemorrhagic Measles.—This form occurs under bad hygienic conditions, and is characterized by a petechial rash, by hemorrhages from the mucous membranes, and by profound prostration.

Complications and Sequelæ.—Bronchopneumonia and acute gastro-intestinal catarrh are the most common complications. Among the less frequent complications or sequelæ may be mentioned membranous or ulcerative laryngitis, otitis, chronic conjunctivitis, pulmonary tuberculosis, cancrum oris, and neuritis.

Diagnosis.—**Rotheln.**—Prodromes are often absent; fever and catarrh are slight; sore throat is marked. The rash appears on the first or second day as a diffuse red blush, or as small, pale-red spots that do not form crescentic-shaped patches; desquamation is scarcely noticeable.

Scarlet Fever.—The onset is more sudden and is marked by vomiting; there is severe sore throat instead of a general catarrh; Koplik's sign is absent, but the tongue is characteristic; the rash appears on the first or second day as a diffuse punctiform erythema; the pulse is out of proportion to the fever, and there is much greater tendency to nephritis.

Prognosis.—Guardedly favorable. Complications are liable to occur and render the prognosis grave.

Treatment.—The child should be quarantined for at least four weeks from the onset of the disease. The sick-room should be well ventilated and moderately darkened. At least two weeks should be spent in bed.

Milk, junket, fruit-juices, broths, eggs, and gruels are suitable forms of nourishment. Water should be proffered at frequent intervals.

Daily inunctions of the body with cold cream or cocoa-butter will serve to allay burning and itching of the skin. When conjunctivitis is marked, the eyes should be protected with dark glasses and frequently cleansed with a solution of boric acid (15 grains to the ounce). Hot baths and hot drinks are indicated when the rash is delayed.

High temperature is best controlled by cold sponging or cold packs. Diarrhea will call for bismuth subnitrate and antiseptics like salol or bismuth-beta-naphthol. When there is severe bronchial catarrh, expectorants with sedatives, like paregoric, are indicated. The following mixture will be found useful:

℞. Potassii citratis . . . . ʒij
Tincturæ opii camphoratæ . . . . . . fʒij–iv
Glycerini . . . . f℥j
Aquæ . . . . q. s. ad f℥iij.—M.
SIG.—A teaspoonful every two hours for a child of three years.

During convalescence tonics—iron, strychnin, and cod-liver oil—are indicated.

RUBELLA.

(Rötheln; German Measles; Epidemic Roseola.)

Definition.—An acute contagious disease resembling both scarlet fever and measles, but differing from these in its short course, slight fever, and freedom from sequelæ.

Etiology.—The disease is highly contagious, and the poison may be carried on clothes or other fomites. It generally occurs in epidemics, but sporadic cases are not uncommon. It is most frequently observed in children, but unprotected adults are not exempt. One attack usually protects from another, but not from measles or scarlet fever.

Period of Incubation.—One to three weeks.

Symptoms.—Prodromes are slight or altogether absent. The disease begins with drowsiness, slight fever, and sore throat. The eruption appears on the first or second day, and varies considerably in its character. In some cases the rash is composed of pale-red, scarcely elevated papules, which are more or less discrete (*rubella morbilliforme*); in others the rash is bright red and diffuse, like that of scarlet fever (*rubella scarlatiniforme*). It begins on the face and rapidly spreads over the entire body, but it fades so rapidly that the face may be clear before the extremities are affected. Slight desquamation frequently follows, though it is often absent. Apart from the sore throat, the catarrhal symptoms are slight. A very constant and somewhat characteristic feature is marked swelling of the postcervical glands.

The duration is from three to five days.

Prognosis.—Good. Complications are very rare.

Treatment is that of measles.

SMALLPOX.

(Variola.)

Definition.—An acute contagious disease characterized by vomiting; lumbar pains; an eruption which is at first papular, then vesicular, and finally pustular; and by fever which is marked by a distinct remission, beginning with the advent of the eruption and lasting until the latter becomes pustular.

Etiology.—The poison of smallpox is extremely tenacious: it may remain latent in clothes or other fomites for a long time, and then be capable of exciting the disease. Unless protected by vaccination or a previous attack, nearly everyone, from the child *in utero* to the aged, is susceptible to the contagion. The colored race seems to be especially predisposed.

Certain protozoa—*Cytorrhyctes variolæ*—which are invariably found in the cells of the vesicles and which present themselves in two forms—one cytoplasmic and the other an intranuclear form—appear to be the specific cause of the disease.

Pathology.—The development of the variolous vesicle is the result of a peculiar degeneration of the protoplasm of the epithelial cells of the rete mucosum. There is a reticular degeneration of the cytoplasm with a more advanced degeneration of the nucleus. Genuine pocks are frequently found in the mouth, esophagus, nose, and larynx. Even the trachea and bronchi may contain them. The spleen is engorged. The organs and muscles reveal fatty and parenchymatous degeneration.

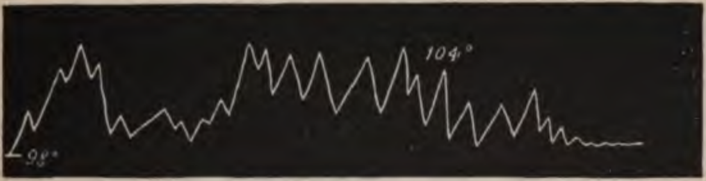

FIG. 17.—Temperature-curve in smallpox.

Varieties.—Discrete; confluent; malignant; varioloid.

Period of Incubation.—Ten days to two weeks.

Symptoms.—**Discrete Smallpox.**—The disease usually begins with a chill or series of chills, followed by vomiting and intense lumbar pains. The fever rises rapidly, reaching its maximum (104°–105° F.) in forty-eight hours, and continues high until the third or fourth day, when it falls several degrees; this remission lasts until the seventh or eighth day—that is, until the time of pustulation—when the temperature again rises. The secondary or suppurative fever shows marked fluctuations; its height is proportionate to the number of pustules; and it falls by lysis about the eighteenth day of the disease. The pulse is full and rapid (120–140); the breathing is hurried; the skin is dry; the bowels are usually constipated, though diarrhea is not uncommon; and the urine is scanty and frequently albuminous.

The Eruption.—About the third or fourth day small red spots are noticed on the forehead, face, and wrists; these are rapidly converted into smooth, round papules that feel like shot under the skin. The eruption rapidly spreads over the entire body. About the third day the papules are converted into clear vesicles, which present a depression or umbilication at their summit. They are also loculated,—*i. e.*, divided into compartments by fibrinous partitions,—so that when pricked with a needle all the contained fluid does not escape. In two or three days the clear fluid becomes turbid and the vesicles are gradually converted into pustules. The latter soon lose the umbilicated appearance. Between the lesions the skin is edematous, so that the body is swollen and the features are unrecognizable. In three days more the pustules dry up, or break and form soft yellow crusts that exhale a peculiar, offensive odor; they adhere to the skin for a week or more. When the scabs fall off, scars or pock-marks generally remain, constituting a permanent deformity.

At the beginning of the disease, before the true variolous eruption appears, either a red blush or a macular rash is often observed on the inner side of the arms and thighs.

Confluent Smallpox.—The papules are abundant and soon coalesce. The extremities are swollen and painful. The secondary fever is very high and irregular. True pocks nearly always develop in the air-passages and give rise to a copious fetid discharge from the nose and throat, to hoarseness, and to cough. Delirium, stupor, and subsultus are frequent symptoms. If the patient recovers, it is after a tedious convalescence, with great facial disfigurement, and often with defective vision or hearing.

Malignant Smallpox.—In some cases the disease is ushered in with high fever, lumbar pains, and great prostration. Soon ecchymoses appear on the skin; bleeding from the mucous membranes follows; and death results before a true variolous rash appears. In other cases the disease advances like ordinary smallpox up to the pustular stage; then the pustules become effused with blood, and bleeding from the mucous membranes follows. This form is also very fatal.

Varioloid.—This is modified smallpox occurring in one who has been partially protected by previous vaccination. The symptoms are mild; the eruption resembles that of common smallpox, but it is usually scant and of short duration; secondary fever is absent.

Complications and Sequelæ.—The most common are bronchopneumonia; pleurisy; inflammations of the eye (ulcerative keratitis, iritis, conjunctivitis); otitis; ulcerative laryngitis; arthritis; and furuncles.

Diagnosis.—**Varicella.**—In this disease prodromes are generally absent and the constitutional symptoms are mild. The eruption appears on the first day; it comes out in successive crops; and, unlike that of smallpox, prefers the covered surfaces. It may be maculopapular at first, but it becomes vesicular within a few hours. The lesions are superficial; vary greatly in size; are usually unilocular, and are rarely umbilicated. Desiccation begins in two or three days, the vesicles becoming irregularly puckered at the periphery and presenting a depressed blackish crust in the center—a highly characteristic appearance. In doubtful cases in children the presence of a typical vaccinal cicatrix constitutes strong presumptive evidence against variola.

Syphilis.—The history of infection; the associated evidences of syphilis (mucous patches, alopecia, etc.); the gradual onset of the illness; the slight fever; the symmetric distribution of the eruption; its dark coppery color; its polymorphous character (papules, vesicles, and pustules associated in a limited area); and the absence of itching will indicate syphilis.

Prognosis.—This depends upon the virulence of the epidemic, the vaccinal condition of the patient, and the amount of eruption. In the discrete cases the prognosis is generally favorable; in the confluent, very grave; and in the malignant, almost hopeless. In the unvaccinated the mortality ranges between 20 and 60 per cent. Among those having one or two typical vaccine scars, the death-rate is very low—usually less than 3 per cent.

Treatment.—The preventive measures against smallpox include the complete isolation of the patient (preferably in a special hospital), the thorough disinfection of all objects that have been in contact with him, and, above all, the vaccination of all who have been or who are likely to be exposed to the contagion. Absolute rest in bed, light bed-clothing, a well-ventilated room of a temperature of 65° F., an easily assimilable but sustaining diet, and the free use of cool drinks are requisites of treatment. The severe lumbar pains will require opium and the application of hot-water bags. Fever is best combated by hydrotherapy—cold sponging, cold packs, or cold baths. Antipyretic drugs should be used with caution.

Gastric irritability may be controlled by diluted hydrocyanic acid (2 minims), subnitrate of bismuth (10 grains), or cocain ($\frac{1}{8}$ grain). When nervous symptoms are not relieved by hydrotherapy, opium with bromids or chloral with bromids should be tried. Alcoholic stimulants are frequently demanded, especially in confluent cases.

An attempt should be made to keep the nasopharynx clean by means of antiseptic sprays or douches. The eyes should also be kept clean by frequent applications of a warm boric-acid solution (15 grains to the ounce).

Prevention of Pitting.—The room should be darkened and the exposed parts covered with cloths wrung out of a weak solution of carbolic acid (1 : 200) or of corrosive sublimate (1 : 5000 or 1 : 10,000). Unfortunately, when the lesions are deeply seated, there are no efficient means of preventing pitting.

In the stage of desiccation, warm baths followed by inunctions with cold cream or olive oil are useful in allaying itching and in hastening the removal of the crusts.

VACCINIA.

(Vaccination; Cow-pox.)

Definition.—A general disease with a local manifestation resembling the pock of variola, and acquired by inoculation with the virus of cow-pox.

History and Object.—The value of vaccination as a means of protection against smallpox was first made known to the world in a paper published by Edward Jenner in 1798.

Recent vaccination gives almost complete immunity against variola; the mortality of smallpox acquired after vaccination is almost inversely proportionate to the number of true vaccine scars.

Etiology.—Vaccinia is induced by inoculating the arm or leg with fresh virus obtained from the udder of a calf suffering from cow-pox (bovine virus). Formerly virus taken from a human vaccine vesicle was also employed (humanized virus), but on account of the risk of transmitting syphilis and other diseases, this source has been practically abandoned.

It has been shown that the addition of glycerin to vaccine lymph serves to preserve it and to free it from pathogenic bacteria.

Time of Performance.—The first vaccination should be performed, as a rule, about the second or third month, the second at the seventh year, and the third at puberty. Vaccination should always be repeated when smallpox is prevalent.

Performance of Vaccination.—The part selected should be thoroughly cleaned with soap and water, then with alcohol, and finally with pure water. A number of cross-scratches should next be made over an area about $\frac{1}{8}$ of an inch in diameter, with a sterilized needle or special scarificator, deep enough to allow of a little oozing of pinkish serum. The virus should then be applied and well rubbed into the exposed lymph-spaces by additional scarification. A shield may be worn for a few hours until the wound has become perfectly dry; after that it should be discarded.

Symptoms.—About the third or fourth day after the operation a papule surrounded by a red areola forms at the seat of inoculation. In two or three days the papule becomes transformed into a clear vesicle with a central depression. The tissues surrounding the vesicle are red, infiltrated, and tender, and the seat of intense itching. The vesicle reaches its full size by the eighth or ninth day, when it ruptures and discharges or dries to a crust. The latter remains for from one to three weeks, when it falls off, leaving a red cicatrix that later becomes white and pitted.

During the course of the eruption there may be slight fever, malaise, restlessness, and enlargement of the axillary glands.

Complications.—Abscess or erysipelas may result from secondary infection. Various generalized eruptions, such as urticaria or erythema multiforme, are occasionally excited by vaccination. Tetanus has occurred in a few instances.

VARICELLA.

(Chicken-pox.)

Definition.—An acute contagious disease of short duration, characterized by slight fever and a discrete vesicular eruption, which disappears in two or three days by desiccation.

Etiology.—The disease occurs sporadically and epidemically. It is observed chiefly in children, but adults are not exempt. One attack usually protects from others. It bears no relation to smallpox.

Period of Incubation.—Fourteen to sixteen days.

Symptoms.—In most cases there is slight fever,—100°–102° F.,—with chilliness and malaise. Not infrequently, however, constitutional symptoms are wholly wanting.

Eruption.—This appears within the first twenty-four hours. At first it is maculopapular, but within a few hours it becomes vesicular. The vesicles are usually sparse; are most abundant upon the trunk; come out in crops; are superficial and very variable in size; are unilocular; and are rarely umbilicated. In two or three days desiccation begins, the vesicles becoming irregularly puckered at the periphery and presenting a depressed blackish crust in the center. The crusts are thin and friable. In the majority of cases there is no marking, but occasionally a few scars or pits remain.

In rare instances gangrene occurs around the vesicles or in other parts (*varicella gangrænosa*).

Diagnosis.—The differential diagnosis between varicella and smallpox has already been considered (see p. 311).

Prognosis.—This is always favorable. Complications are very rare.

Treatment.—No special treatment is required. The child should be separated from others until the crusts have disappeared. Itching may be allayed by applications of carbolized vaselin.

DIPHTHERIA.

Definition.—An acute contagious disease excited by the Klebs-Löffler bacillus, and characterized by moderate fever, glandular enlargement, great prostration, anemia, and the formation of a false membrane upon certain mucous membranes, especially those of the throat and adjacent parts.

Etiology.—Three-fourths of the cases occur in children before the tenth year. Damp, cold weather and bad hygienic surroundings favor outbreaks of the disease.

Chronic catarrhal affections of the nose and throat distinctly increase the susceptibility to infection. The immunity afforded by one attack is of short duration.

The exciting cause is the Klebs-Löffler bacillus,—the Bacillus diphtheriæ,—which is found chiefly in the affected mucous membranes, and only exceptionally in the blood and in distant organs. The constitutional symptoms are caused by the absorption of toxins produced by the bacillus.

Pathology.—The false membrane is usually found on the tonsils, pillars, and pharynx, but it may extend to the mouth, larynx, or nose. The bacillus coming in contact with the mucous membrane causes a coagulation-necrosis of the superficial cells and an inflammatory exudation, the whole constituting the false membrane. The latter usually has a grayish or yellowish appearance, is firmly attached to the underlying tissues, and when forcibly removed, leaves a raw and bleeding surface. Microscopically, it is composed of fibrin, epithelial cells, and leukocytes (more or less degenerated), Klebs-Löffler bacilli, and pyogenic cocci.

The lymphatic glands near the seat of infection are swollen. Focal necrosis, due to the action of the toxin, is found in the liver and other organs. The heart, kidneys, and liver are the seat of fatty and parenchymatous degeneration. Interstitial hemorrhages are frequently observed and are the result of hyaline degeneration of the capillary walls and thrombotic obstruction. Such lesions as congestion, edema, bronchopneumonia, and atelectasis are frequently encountered in the lungs.

Types.—Diphtheria may be divided according to the location of the exudate into: (1) Faucial; (2) laryngeal; and (3) nasal. According to the severity of the attack it may be divided into: (1) Mild; (2) grave; (3) malignant.

Period of Incubation.—Two days to a week.

Symptoms.—**Faucial Diphtheria.**—The disease commonly begins with chills, moderate fever, malaise, and sore throat. The fever, as a rule, is not very high (102°–104° F.), and its course is quite irregular. The pulse is rapid and feeble; the bowels are constipated; the urine is scanty and frequently albuminous; and the prostration and pallor are often out of all proportion to the severity of the febrile symptoms.

Local Phenomena.—The child complains of difficult swallowing; the muscles of the neck feel stiff; there is tenderness under the jaw; the lymphatic glands are considerably swollen; and the tonsils, faucial pillars, uvula, and posterior pharyngeal wall are covered with a grayish-white membrane which, when stripped off, exposes a raw bleeding surface. The membrane may spread to the nose or larynx.

The course of the disease is indefinite, but the average duration is from one to two weeks.

Laryngeal Diphtheria.—This is usually secondary by extension from the fauces, but it is occasionally primary. It is recognized by hoarseness or aphonia, croupy cough, progressive dyspnea, and stridulous breathing. The alæ of the nose play; the sternocleidomastoids are prominent; the suprasternal notch is deepened; and the base of the chest is retracted. Shreds of false membrane are sometimes expectorated in the violent fits of coughing. The pulse is rapid and feeble, but the temperature is rarely high. Death often results from suffocation, but recovery is not impossible even in the most unpromising cases.

Nasal Diphtheria.—This is usually secondary. It is characterized by grave constitutional symptoms—high fever, marked glandular involvement, and great prostration; by an offensive discharge from the nose; by epistaxis; and by excoriation of the lips. The false membrane may be detected on inspection.

Complications and Sequelæ.—The most common complications are heart failure from myocarditis or neuritis of the cardiac nerves, bronchopneumonia, acute nephritis, hemorrhage from the ulcerated surfaces, and otitis media. The most important sequel is *postdiphtheric paralysis.* This generally occurs during convalescence, and is observed in about 15 per cent. of all cases. There is no relation between the severity of the attack of diphtheria and the liability to paralysis; mild cases, which are thought to be simple pharyngitis, being sometimes followed by troublesome paralysis. The pharynx is the most common seat, and the palsy is recognized by difficult swallowing and the regurgitation of liquids through the nose. Next in frequency the eyes are involved, and strabismus or ptosis develops. The heart may be affected, and if sudden death does not result, the condition may be manifested by tachycardia or bradycardia. In some instances there is an extensive involvement of the extremities. The paralysis is due to toxic neuritis.

Diagnosis.—**Scarlet Fever.**—This can be distinguished by the characteristic strawberry tongue, the very rapid pulse, the diffuse punctiform rash, and the absence of the diphtheria bacillus.

Follicular Tonsillitis.—The differential diagnosis between this disease and diphtheria has already been considered (see p. 32).

Prognosis.—This must always be guarded. The disease is very fatal during the first two years of life. The average mortality at the present time ranges between 15 and 20 per cent. The nasal and laryngeal forms are always grave. Death may be due to exhaustion from the toxemia, involvement of the larynx, bronchopneumonia, cardiac paralysis, or nephritis.

Treatment.—**Prophylaxis.**—As diphtheria is prone to attack unhealthy mucous membranes, nasopharyngeal catarrh in children should receive careful attention. Large tonsils and adenoid growths should be removed. Those who have been exposed to the contagion should receive immunizing doses of antitoxin (500 units). Patients with diphtheria should be kept isolated until their throats are free from virulent bacilli. The bedroom, bedding, clothing, and all utensils used by the sick should be thoroughly disinfected.

Treatment of the Attack.—The sick-room should be well ventilated, and the temperature maintained at about 70° F. It is desirable to have the atmosphere moist, and this may be accomplished by generating steam in an ordinary kettle or in a steam atomizer, or by slaking large quantities of quicklime in the room. Young children, especially when laryngeal symptoms are present, are best treated in a steam-moistened tent. Absolute rest must be enforced. The diet should be of the most nutritious and easily digested character. Cool water should be given freely. Antitoxin should be administered in every case at the earliest possible moment. In pharyngeal cases the initial dose should be from 3000 to 4000 units. If no decided improvement follows within twelve hours, the dose should be repeated. Laryngeal cases require from 6000 to 8000 units. The injections may be made into the loose subcutaneous tissue of the pectoral region, side of the abdomen, or interscapular space. Strict antiseptic precautions should be taken in the operation.

Apart from antitoxin, the most important remedies are those which tend to maintain the bodily strength. Alcoholic stimulants are usually indicated, especially in the late stage of the disease. In septic cases alcohol is particularly well borne, a child of three years often being able to take several ounces of whisky a day with advantage. Next to alcohol, strychnin ($\frac{1}{120}$ grain every three or four hours) is the best stimulant. In profound adynamia, digitalis, caffein, camphor, and musk are also useful.

Local treatment is often useful. When, however, the applications cause violent struggling and exhaust the child, it is better to desist. Irrigation of the nose and pharynx may be practised every two or three hours by means of a soft catheter attached to a fountain syringe. The solutions most useful for the purpose are warm normal salt solution or a warm saturated solution of boric acid. A solution of hydrogen dioxid (1 to 3 of lime-water) or the following solution of Löffler may be applied to the throat by means of a swab:

℞.	Mentholis	ʒiiss
	Toluolis	fʒx
	Alcoholis absoluti	f℥ij
	Liquoris ferri chloridi	fʒj.—M.

Externally, hot or cold applications, whichever may be the more agreeable, are useful in relieving pain and soreness in the throat. In laryngeal cases tracheotomy or intubation, preferably the latter, should not be deferred when dyspnea becomes urgent.

Convalescence must be managed with special care on account of the tendency to sudden heart-failure. Anemia will require plenty of nourishing food, and such remedies as iron, arsenic, and cod-liver oil. Paralysis usually yields to strychnin, combined with applications of massage and electricity.

ERYSIPELAS.

Definition.—An acute infectious disease excited by the Streptococcus pyogenes, and characterized by high fever and a peculiar inflammation of the skin and subcutaneous tissues.

Etiology.—The disease is somewhat contagious, and the poison can be carried in fomites. Certain families and certain individuals seem particularly predisposed. Puerperal women and wounded persons are very susceptible. Diseases which lower the vitality, especially Bright's disease, predispose. One attack does not protect against a recurrence, but rather favors it. Erysipelas was formerly divided into traumatic and idiopathic varieties; but the two are identical, and it is probable that in those cases in which there is no conspicuous wound there is a slight abrasion through which the poison gains admittance.

The *exciting cause* is the Streptococcus pyogenes.

Pathology.—Erysipelas most frequently manifests itself on the face. The part is bright red in color, swollen, indurated, and sharply circumscribed. The various strata of the skin are infiltrated with serum, and leukocytes and streptococci are found in the lymph-spaces. In severe cases the inflammatory products are converted into pus, and abscesses form.

Period of Incubation.—Three to seven days.

Symptoms.—Prodromes are sometimes present, and consist of slight fever, chilliness, malaise, and tingling of the part to be affected. In many cases the disease is ushered in suddenly with a chill, followed by pain in the head and limbs and a high, irregular fever. The temperature may reach 104° or 105° F. in twelve or twenty-four hours. The pulse is full and rapid; the tongue is heavily coated; the appetite is lost; the bowels are constipated; and the urine is scanty and often slightly albuminous. There is usually a marked leukocytosis.

Local Phenomena.—The inflammation usually begins in the neighborhood of the nose, and spreads upward and laterally over the head to the neck, where it frequently stops. The affected part has a crimson hue; it is swollen and tense, and frequently ends in a sharply defined ridge, beyond which, however, projections can be felt advancing into the subcutaneous tissue. The surface of the inflamed patch is at first smooth and glazed, but later it is covered with minute vesicles or blebs. The patient complains of burning and tingling; the surrounding parts are extremely edematous, so that the features may be scarcely recognizable. In four or five days the redness begins to fade and the swelling to subside; desquamation follows; the general symptoms improve; and the fever falls by crisis. The average duration is from a week to ten days. Relapses are extremely common.

Erysipelas Ambulans.—Sometimes the inflammation disappears in one place and reappears in another, and so continues indefinitely. In such cases typhoid symptoms, such as muttering delirium, a brown, fissured tongue, and subsultus tendinum, develop.

Complications.—These are not very common. Septicemia, ulcerative endocarditis, nephritis, acute rheumatism, edema of the larynx, pneumonia, and meningitis are occasionally seen.

Diagnosis.—**Erythema.**—The absence of high fever, of marked swelling, and of an abrupt ridge will serve to distinguish erythema from erysipelas.

Acute Eczema.—The swelling is less marked; the itching is intense; the swelling and redness are not circumscribed, but shade gradually into healthy tissue; and there is no fever.

Prognosis.—In the robust the prognosis is favorable. In the old, in alcoholic subjects, and in those suffering from chronic nephritis the prognosis must be guarded. Ambulatory erysipelas may kill by exhaustion.

Treatment.—As in other contagious diseases, isolation and disinfection are the most important prophylactic measures. Especially necessary is it to guard parturient and surgical patients from the contagion.

A supporting liquid diet should be given. Alcoholic stimulants are sometimes required in considerable quantities. High fever is best controlled by cold sponging or the cold pack. Restlessness, delirium, and insomnia will call for applications of ice to the head, and perhaps the administration of morphin, chloral, or bromids.

Of the numerous special remedies recommended for erysipelas, the one which has enjoyed the most favor is the tincture of ferric chlorid (15 to 30 minims every three hours).

Local Treatment.—Among the numerous local applications recommended may be mentioned: Lotions of lead-water and laudanum, of carbolic acid (1 : 40), of picric acid (1 : 100), and of sodium salicylate (1 : 20). In the hands of the author ointments of ichthyol (20 per cent.) and of soluble silver (unguentum Credé) have proved most satisfactory.

The following combination often acts extremely well:

℞.	Ichthyol	gr. xxx
	Resorcinolis	ʒss
	Unguenti hydrargyri	ʒiv
	Adipis lanæ hydrosi	ʒv.—M.

(ROSWELL PARK.)

Local abscesses should be incised and treated antiseptically. Extension to the nose and throat will call for antiseptic sprays or washes.

INFLUENZA.

(La Grippe; Catarrhal Fever; Epidemic Catarrh.)

Definition.—An acute infectious disease characterized by fever, marked prostration, severe muscular pains, and catarrhal inflammation of certain mucous membranes, especially those of the respiratory tract.

Etiology.—The disease occurs in epidemics that usually have their origin in Russia, whence they spread with wonderful rapidity over both continents. The exciting cause is the extremely small, non-motile bacillus discovered by Pfeiffer in 1892. It is readily obtained from the sputum. When prevalent, no age and neither sex is exempt. One attack does not confer immunity against others.

Pathology.—Influenza does not often kill except by its complications. The latter are most frequently associated with the respiratory tract, and consist chiefly of catarrhal pneumonia, croupous pneumonia, and pleurisy.

Symptoms.—The disease begins abruptly with lassitude, malaise, chilliness, severe pain in the head and back, fever ranging between 102° and 104° F., and extreme prostration, which is out of proportion to the fever and any existing local inflammation. The catarrhal symptoms are injection of the eyes, sneezing, hoarseness, and hard paroxysmal cough. In simple cases the temperature falls in three or four days by crisis, but complications not infrequently prolong the case for several weeks.

In some cases the catarrh of the respiratory tract is the chief feature; in others the gastro-intestinal tract is attacked, and the symptoms resemble cholera morbus; in a third group neuralgic pains in the head, back, and limbs are the most striking phenomena.

Complications and Sequelæ.—The most important are: Catarrhal pneumonia, croupous pneumonia, pleurisy, neuritis, cardiac neuroses, and pericarditis. Permanent insanity is an occasional sequel.

Diagnosis.—**Acute Bronchitis.**—The fever is not so high; there is little or no prostration; and the pains in the head and back are not nearly so marked as in influenza.

Typhoid Fever.—The gradual onset, typical temperature-curve, epistaxis, diarrhea, Widal reaction, and rash will indicate typhoid fever.

Prognosis.—Uncomplicated cases nearly always recover. In the very old and in those debilitated by chronic disease influenza not infrequently proves fatal.

Treatment.—Hygienic measures are of the first importance; these include immediate and absolute rest in bed, a carefully selected diet, pure air without draft, and attentive nursing. Complications and relapses can generally be traced to a neglect of these rules.

In mild cases a hot foot-bath, some mild refrigerant, such as spirit of nitrous ether or solution of ammonium acetate, and at night a dose of Dover's powder (5 to 10 grains) will usually suffice. If there be constipation, a few fractional doses of calomel may be given with advantage.

In more severe cases quinin (2 to 5 grains thrice daily) may be given throughout the attack. Pains are controlled to some extent by phenacetin with salicylates or benzoates.

The following combination is often useful:

℞. Acetphenetidini
Salophen
Sodii benzoatis . . . . āā ʒj.—M.
Fiant chartulæ No. xij.
SIG.—One every three or four hours.

When the suffering is intense, morphin should be used hypodermically. Violent headache is treated best by small doses of phenacetin and the application of an ice-cap to the head.

Heart-failure should be combated by alcohol and strychnin. Bronchial catarrh will require the remedies indicated in simple bronchitis. Sleep may be induced by opium, sulphonal, or chloralamid.

MUMPS.

(Epidemic Parotitis.)

Definition.—An acute contagious disease, characterized by inflammation of the parotid and other salivary glands.

Etiology.—The disease occurs sporadically and epidemically. It is most frequently observed in young children, but unprotected adults are not exempt. Males are more susceptible than females. The disease is highly contagious, and the virus is probably contained in the saliva, but it has not been isolated. One attack confers immunity against others.

Pathology.—As the disease is so seldom fatal, very little opportunity is afforded for studying its intimate pathology. The parotid glands are the seat of an inflammatory infiltration, but suppuration very rarely occurs. In males the inflammation shows a marked tendency to leave the parotids and to involve the testicles. In girls, transference of the inflammation to the ovary, vulva, or mammary gland is occasionally seen.

Period of Incubation.—One to three weeks.

Symptoms.—The disease is ushered in with chilliness, malaise, and moderate fever (101°–103° F.), followed by swelling of one parotid gland. The swelling is observed below and in front of the ear, is pyriform in shape, and has a doughy feel. The surrounding tissues are edematous, the submaxillary glands are often swollen, and the features may be distorted beyond recognition. The movements of the jaw are restricted and painful. The saliva is usually much diminished, but occasionally it is increased. In most cases the other parotid becomes similarly affected. The duration of the disease is from five to seven days.

Complications.—Orchitis is the most important complication. It is usually seen in adolescence; in childhood it is very rare. Atrophy of the testicle sometimes follows. Deafness, nephritis, suppuration of the gland, and pneumonia are rarely encountered.

Prognosis.—Favorable.

Treatment.—The patient should be kept in bed. Isolation should last three weeks from the onset of the disease. Mild aperients and refrigerants are useful. When the pain is severe, hot fomentations containing laudanum prove soothing. In mild cases, covering the gland with cotton batting will suffice.

Orchitis will require rest, suspension of the affected gland, and the application of lead-water and laudanum or, better still, of an ointment of guaiacol (10 per cent.). After the tenderness has subsided, an ointment of mercury and belladonna will be found useful in reducing the swelling.

YELLOW FEVER.

Definition.—An acute infectious, endemic or epidemic disease, characterized by fever of one or two paroxysms, jaundice, albuminuria, and a marked tendency to hemorrhage, especially from the stomach.

Etiology.—The specific organism of yellow fever has not yet been isolated. Man is inoculated through the bites of a certain species of mosquito,—*Stegomyia calopus*,—which serves as the intermediate host for the parasite. The mosquito is infected only by biting a yellow-fever patient during the first three days of the disease and cannot transmit the infection until a period of from twelve to twenty days has elapsed. The disease is not conveyed by fomites.

Yellow fever occurs endemically in tropical sea-ports, whence it occasionally spreads to temperate zones. The predisposing factors are those which are favorable to the growth of mosquitos—high temperature, surface drainage, and swampy soil. The colored race is less susceptible than the white. Strangers in an infected district are more liable to be attacked than residents. One attack usually confers immunity from others.

Pathology.—The tissues are stained yellow. The liver presents a reddish-yellow (autumn-leaf) mottled hue, and is the seat of extensive fatty degeneration. The kidneys usually show the lesions of acute hemorrhagic nephritis. The gastro-intestinal mucous membrane is swollen, congested, and frequently infiltrated with blood. The heart-muscle is pale and fatty.

Period of Incubation.—Three to four days.

Symptoms.—*First Stage.*—The disease begins with a chill, followed by pain in the head, back, and limbs. The temperature rises rapidly until it reaches its maximum (103°–105° F.).

The pulse is at first accelerated, but as the temperature rises it shows a marked tendency to fall, sometimes dropping in grave cases to 80 or even to 70 a minute by the third day. The face is flushed; the conjunctivæ are injected; the pupils are small; the tongue is coated; the epigastrium is tender; the stomach is irritable and unretentive; the bowels are constipated; and the urine is scanty and often albuminous by the end of the first day.

Jaundice is rarely marked before the second or third day, although a slight icteroid tinge of the conjunctivæ is often noticeable within the first twenty-four hours. The first stage usually lasts from three to five days, and is followed by a rapid fall in the temperature and an improvement in all the symptoms (stage of calm or remission). At this time convalescence may begin or the patient may pass into the second stage.

The *second stage* is characterized by deep jaundice, persistent vomiting, vomiting of dark blood (*black vomit*), marked albuminuria, and often by suppression of urine and hemorrhages from the mucous surfaces. The mind usually remains clear until very near the close, but in some cases delirium and stupor develop. This stage may be afebrile, but not infrequently the temperature rises again after the period of calm, while the pulse remains extremely low (50 to 40 a minute). Death usually results from collapse or uremia. The duration of the disease is from three to ten days.

Diagnosis.—**Dengue.**—This disease does not exhibit a slow pulse with a rising temperature, early albuminuria, jaundice, or black vomit.

Acute Yellow Atrophy of the Liver.—The rapid pulse, the diminution in the size of the liver, the slight fever, the marked cerebral symptoms, and the presence of leucin and tyrosin in the urine will indicate acute yellow atrophy.

Remittent Fever.—This may be distinguished by the enlargement of the spleen, the multiple remissions, the presence in the blood of the hematozoa of Laveran, and by the absence of black vomit.

Prognosis.—Always grave. The average mortality in different epidemics is from 20 to 70 per cent. In individual cases high fever, a very slow pulse, marked cerebral symptoms, black vomit, and suppression of urine are unfavorable features.

Treatment.—"The spread of yellow fever can be most effectually controlled by measures directed to the destruction of mosquitos and the protection of the sick from the bites of these insects."[1]

Absolute rest in a quiet, well-ventilated room and careful nursing are essential. Only the blandest food should be allowed. Many clinicians of wide experience advocate the withholding of all food during the first day or two. For the gastric irritability a sinapism may be applied to the epigastrium, and cracked ice, champagne, hydrocyanic acid, or cocain may be given internally. Fever is best controlled by the external application of cold. Suppression of urine will call for dry cupping over the loins, alkaline diuretics, hot-air baths, and subcutaneous or rectal injections of warm saline solutions. Remedies have little effect upon the black vomit. Tincture of ferric chlorid, adrenalin solution (1 : 1000), and oil of turpentine have been recommended.

ACUTE GENERAL TUBERCULOSIS.

(**Acute Miliary Tuberculosis.**)

Definition.—An acute infectious disease excited by the tubercle bacillus, and characterized anatomically by the simultaneous formation of miliary tubercles in many parts of the body.

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[1] Report of U. S. Army Commission, *Jour. Hyg.*, vol. ii., No. 2.

Etiology.—The disease usually develops in early adult life. Certain infectious diseases, like measles and whooping-cough, seem to predispose. General tuberculosis is almost always secondary to local tuberculosis—pulmonary phthisis or a scrofulous lymphatic gland. The bacilli are probably disseminated by the veins.

Pathology.—All the organs may be uniformly infiltrated with discrete tubercles, but more commonly certain organs, like the brain and lungs, are more affected than others.

Symptoms.—The onset is gradual and characterized by anorexia, malaise, headache, increasing prostration, and fever. The temperature is moderately high (102°–104° F.), very irregular, and marked by evening exacerbations and morning remissions. The respirations are hurried and the pulse is rapid (140 to 150) and feeble. Cough may or may not be present. As the disease advances typhoid symptoms develop —brown, fissured tongue, muttering delirium, subsultus tendinum, carphologia, and stupor. Tubercle bacilli are rarely found in the sputum or in the blood.

When the lungs are chiefly affected, there are: Dyspnea, rapid breathing (40 to 60 a minute), hard cough, mucopurulent and bloody expectoration, and cyanosis. Signs of consolidation can rarely be elicited, but auscultation usually reveals sibilant and moist râles.

When the meninges are chiefly affected, there are: Intense headache, convulsive seizures, photophobia, delirium, facial palsies, stupor, coma, and Cheyne-Stokes breathing. Tubercles may occasionally be detected on the retina.

When the intestines and peritoneum are affected, there are: Pain, tenderness, abdominal distention, and diarrhea.

Prognosis.—The disease is always fatal. The duration is from three to eight weeks.

Diagnosis.—The disease closely resembles **typhoid fever,** and there is no doubt that the mortality of the latter is enhanced by included cases of unsuspected general tuberculosis.

TYPHOID FEVER.	ACUTE GENERAL TUBERCULOSIS.
Epistaxis is common.	Infrequent.
Temperature runs a regular course.	Temperature runs a very irregular course.
Diarrhea is frequent.	Infrequent.
A roseolar eruption is generally present.	Rarely present.
Respirations are rapid.	Usually much more rapid.
Pulse is rapid.	Usually much more rapid.
Cyanosis rarely marked.	Often distinct.
Facial palsies are absent.	Are occasionally noticeable.
Widal reaction is present.	Is absent.

Treatment.—This is purely palliative. The diet should consist of milk, eggs, and broths. Stimulants are required. Fever should be controlled by cold sponging or small doses of phenacetin. Severe cough and insomnia will call for morphin.

WHOOPING-COUGH.

(Pertussis.)

Definition.—An infectious disease, characterized by catarrh of the respiratory tract and peculiar paroxysms of cough ending in prolonged crowing or whooping inspiration.

Etiology.—The disease occurs both sporadically and epidemically. It is most frequently met with in children, but unprotected adults are not exempt. The disease is unquestionably contagious, and the virus seems to be associated with the sputum. One attack protects from others.

Pathology.—No characteristic lesions are observed after death. The poison excites an inflammation of the respiratory mucous membrane, and probably irritates the peripheral filaments of the pneumogastric nerve, and so causes the paroxysmal cough. In fatal cases pulmonary complications are usually discovered, such as catarrhal pneumonia, pulmonary collapse, and emphysema.

Symptoms.—There are three stages: (1) The catarrhal stage; (2) the paroxysmal stage; and (3) the stage of decline.

Catarrhal Stage.—The disease begins with the symptoms of coryza and bronchial catarrh,—slight fever, sneezing, running from the nose, dry cough, and râles,—but it does not respond to the ordinary remedies for catarrh, and after lasting one or two weeks passes into the paroxysmal stage.

Paroxysmal Stage.—The cough becomes more violent and paroxysmal. During the paroxysm the face is cyanosed, the eyes are injected, and the veins distended. The cough frequently induces vomiting, and, in severe cases, epistaxis or other hemorrhages. The close of the paroxysm is marked by a long-drawn, shrill, whooping inspiration due to the spasmodic closure of the glottis.

The number of paroxysms, or "kinks," varies from ten or twelve to forty or fifty in the twenty-four hours. From the forcible propulsion of the tongue against the lower incisors, an ulcer is frequently formed on the frenum. The duration of this stage is three or four weeks.

Stage of Decline.—The paroxysms grow less frequent and less violent and finally cease. Protracted cases are followed by anemia and prostration.

Duration.—The entire duration of the disease is from a few weeks to four months.

Complications and Sequelæ.—The chief are broncho-pneumonia, collapse of the lung, acute emphysema, and hemorrhage from the nose or into the conjunctiva. Paralysis from meningeal hemorrhage occasionally occurs. Severe cases are sometimes followed by cancrum oris, chronic bronchitis, or tuberculosis.

Treatment.—Prophylaxis consists in isolation of the patient and the thorough disinfection of all articles that have been used by him. Quarantine should last until the cough ceases.

Fresh air, sunlight, protection from changes of weather, and a light but nutritious diet are essential. In some cases it may be desirable to keep the patient in his room, or even in bed, for the first few days, but ordinarily, if the weather is good, he need not be confined indoors. In advanced cases sea-air often acts most favorably.

Of the many special remedies advocated, those most worthy of confidence are belladonna (in ascending doses until constitutional effect is produced), antipyrin (1 grain every two hours at one year of age), quinin (10 grains a day at three years of age), bromoform (1 to 2 minims at two years of age), and sodium bromid (3 to 5 grains every three hours at two years of age). Chloral (3 grains at two years of age) may be given in severe cases at bedtime to secure sleep. Such a combination as the following is often useful:

℞.	Sodii bromidi	gr. l
	Antipyrinæ	gr. xv
	Glycerini	f℥ss
	Aquæ menthæ piperitæ . . . q. s. ad	f℥iij.—M.

SIG.—A teaspoonful every two hours for a child one year old.

Antiseptic and sedative sprays, when feasible, sometimes afford much relief; the best are the solution of hydrogen dioxid (1 to 6), menthol (5 per cent. in liquid paraffin), and resorcin (1 per cent. aqueous solution).

The child must be carefully guarded during convalescence, on account of the great danger of catarrhal pneumonia. Tonics, especially quinin, iron, and cod-liver oil, are very useful at this period.

CHOLERA.

(Asiatic Cholera; Epidemic Cholera; Malignant Cholera.)

Definition.—An acute infectious disease, generally epidemic, excited by Koch's comma-bacillus, and characterized by vomiting and purging of serous material, painful cramps, and collapse.

Etiology.—Cholera is constantly present in certain parts of India, and under favorable conditions is carried thence to other parts of the world. The exciting cause is the comma-bacillus of Koch, a short, slightly curved, motile rod with a single flagellum. This organism is found abundantly in the intestinal discharges of choleraic patients. Outside the body its growth is favored by heat and decomposing animal matter.

The disease always spreads along the lines of traffic, hence epidemics nearly always begin at the sea-coast and extend inland. Cholera is slightly, if at all, contagious; like typhoid fever, the poison is not carried through air, but chiefly through drinking-water and food. Flies are undoubtedly important factors in conveying the germs to food. Laundresses and nurses, from their contact with the evacuations, readily acquire the disease. Epidemics are more frequent in summer than in winter. No age is exempt, but the old are more susceptible than the young. The intemperate, the debilitated, and those suffering with gastro-intestinal catarrh are especially predisposed.

Pathology.—The body is shriveled; movements of the corpse are sometimes observed; rigor mortis is marked and prolonged. The tissues are dry, and the large veins and right side of the heart contain thick, dark blood. The serous cavities are empty and their surfaces sticky. The intestines contain more or less rice-water fluid, from which cultures of bacilli can be made.

The mucous membrane has a pinkish color and is often the seat of ecchymoses; the solitary and Peyer's glands are swollen. Frequently extensive desquamation of the epithelial lining is observed. The liver and kidneys are the seat of acute parenchymatous degeneration.

The symptoms of cholera are doubtless due to the absorption of poisonous substances elaborated by the bacilli in the intestines.

Period of Incubation.—Three to five days.

Symptoms.—The severity of the symptoms varies considerably. In well-marked but favorable cases there are three stages: (1) Invasion; (2) algid or collapse; (3) reaction.

Stage of Invasion.—The disease usually begins with malaise, headache, diarrhea, rumbling noises in the intestines, and colic. Frequently these symptoms continue a few days and then subside; such cases are termed *cholerine*, and are as infectious as the fully developed disease.

Stage of Collapse.—The diarrhea grows more marked; the evacuations become copious, lose their feculent character, assume a rice-water appearance, and are discharged forcibly but without pain. Vomiting soon develops, and the ejected material resembles that passed by the bowels. Thirst is unquenchable. Severe cramps seize the muscles of the calves of the legs, thighs, arms, and abdomen. The surface is cold and covered with a clammy sweat; the breath is cool; the temperature in the axilla ranges from 95° to 85° F., while in the rectum it may rise to 103° F. or more. The voice is husky and finally reduced to a whisper; the respirations are quickened; the pulse becomes more and more feeble; the body is livid and shriveled; the hands resemble those of a washerwoman; the features are pinched and sometimes distorted; the eyes are frightfully sunken. The urine is more or less suppressed, and the little that is passed generally contains albumin and sugar. Consciousness is usually retained until near the end, when coma sets in.

The duration of this stage is from a few hours to two days.

Stage of Reaction.—Sometimes, even when death seems imminent, the surface temperature begins to rise; the urine increases; the pulse strengthens; the vomiting ceases; the evacuations from the bowels become less frequent and begin to assume a feculent character, and convalescence is ultimately established.

Occasionally, instead of convalescence, symptoms of a typhoid type develop, such as moderate fever, a brown, fissured tongue, subsultus, muttering delirium, and coma. This condition, which is generally fatal, has been regarded as uremic.

Cholera Sicca.—In very violent cases collapse and death may follow without there having been any evacuation. After death the intestines contain rice-water fluid, which was not discharged during life probably on account of paralysis of the muscular coat of the bowel.

Complications and Sequelæ.—The chief complications are: nephritis, pneumonia, pleurisy, parotitis, ulceration of the cornea, diphtheric inflammation of the throat and fauces, abscesses, and local gangrene.

Diagnosis.—The differential diagnosis between Asiatic cholera and **cholera morbus** has already been considered (see p. 77).

Prognosis.—This depends largely upon the type. The mortality averages about 50 per cent. In the old, very young, debilitated, and intemperate the disease is very fatal.

In individual cases early collapse and a low surface temperature are unfavorable conditions.

Treatment.—Personal prophylactic measures against the disease include removal from the infected districts, restriction of the diet to bland, easily digested food, thorough sterilization of drinking-water and milk, the protection of all food from contamination by flies and other insects, the avoidance of overwork, exposure to wet and cold, and undue excitement, and the prompt treatment of any gastro-intestinal disturbance that may arise. Certain acids, especially sulphuric acid, have long been advocated as preventives of cholera. Finally, vaccination with attenuated cholera cultures, as practised by Haffkine in India, has given encouraging results.

Precautionary measures pertaining to the sick comprise isolation, absolute cleanliness, and the thorough disinfection of excreta, soiled clothing, etc.

The medicinal treatment of cholera resolves itself into that of the prodromal stage, that of the algid stage, and that of the reaction stage.

Prodromal Stage.—From the first appearance of diarrhea the patient should go to bed and remain there. Food should be withheld. If there be a history of indigestible food having been taken, a laxative dose of calomel should be given; otherwise, aperients should be avoided. Hot stupes may be applied to the abdomen. If there is much colic, morphin may be given hypodermically. For the diarrhea, bismuth subnitrate is perhaps the best astringent.

Algid Stage.—Intravenous injections of warm saline solutions undoubtedly afford the best means of combating the anhydremia and of restoring the failing circulation. Rectal injections of hot tannic solutions (2 per cent.), as strongly recommended by Cantani, may also be used. The body-temperature should be maintained by hot applications or hot baths. Diffusible stimulants, like ether and camphor, may be given hypodermically.

To allay thirst, ice or iced Seltzer water may be given at frequent intervals. The painful cramps are best treated by warm applications, hot baths, gentle friction with anodyne liniments, and, above all, by intermittent chloroform inhalations. In suppression of urine the most promising measures are dry cupping over the loins and rectal and intravenous injections of saline solutions.

Reaction Stage.—In this stage liquid foods in small quantities are permissible. Milk with lime-water, whey, thin gruels, albumin-water, and light broths are the most appropriate. The return to ordinary food should be effected most gradually.

TETANUS.

(Lockjaw.)

Definition.—An acute infectious disease excited by a special bacillus, and characterized by painful tonic spasms of the voluntary muscles.

Ætiology.—The exciting cause is the Bacillus tetani, a motile, spore-bearing, anaërobic rod, multiplying in garden-earth, street dirt, and the intestinal discharges of herbivorous animals. The disease is contracted through infection of wounds with matters containing the bacillus or its spores. Lacerated and punctured wounds about the soles of the feet and palms of the hands are especially liable to become infected. Occasionally no history of injury is obtainable. The colored race appears to be particularly vulnerable.

Pathology.—There are no characteristic lesions. Congestion of the spinal cord and of the nerves leading to the wound is sometimes seen. The bacillus produces, at the point of inoculation, an intensely virulent poison, which, being absorbed, vents itself in a special manner upon the central nervous system.

Period of Incubation.—From a few days to three or four weeks.

Symptoms.—The disease begins with a feeling of rigidity in the muscles of the neck and lower jaw; by degrees the muscles of the back, abdomen, and lower extremities are similarly involved. The brow is wrinkled, the corners of the mouth are drawn upward (*risus sardonicus*), the jaws are tightly closed (*trismus*), and the body becomes arched, the patient resting on his head and heels (*opisthotonos*). There is extreme hyperesthesia, so that the slightest touch causes a violent exacerbation of the spasm, which is attended by excruciating pain. If the respiratory muscles are involved, there is intense dyspnea. The temperature is variable. It is usually elevated during the paroxysms and just before death it may rise to 107° F. or more. The mind is clear to the end. The duration is from a few days to several weeks.

Prognosis.—In acute cases the prognosis is very grave death usually resulting within a week from heart-failure, asthenia, or asphyxia. Cases developing after a long period of incubation and not characterized by violent seizures not infrequently end in recovery.

Treatment.—The wound should be enlarged, freed from all foreign matter, and treated with some active antiseptic. The most hopeful means of neutralizing the toxin already absorbed is the prompt injection of tetanus antitoxin in large doses. The drugs most effective in subduing the convulsions are the bromids and chloral. These should be given in large doses. Morphin and eserin are useful adjuvants. Inhalations of chloroform or of amyl nitrite afford temporary relief. The patient should be kept absolutely quiet and protected from cold. The administration of nutriment in liberal quantities is of the utmost importance. Alcohol is often necessary.

DENGUE.

(Breakbone Fever; Dandy Fever.)

Definition.—An acute infectious disease, characterized by pains in the muscles and joints, a variable rash, and a febrile course of two paroxysms.

Etiology.—Dengue is confined almost entirely to hot climates. Although it occurs in epidemics, its contagiousness is still a matter of dispute.

Period of Incubation.—Three to five days.

Symptoms.—The invasion is usually sudden, and is attended with lassitude, chilliness, headache, intense pain in the muscles and joints, and high fever. The latter rises rapidly, often reaching a maximum of 104° to 105° F. in a few hours. The pulse is rapid and full; the respirations are accelerated; the mind is often delirious; the urine is scanty; the superficial lymph-glands are enlarged; the joints are painful, tender, and swollen. In two or three days the temperature falls, and an afebrile period follows in which the patient is free from pain, but is profoundly prostrated. During the remission a roseolar or a diffuse erythematous rash generally appears; this lasts two or three days and is followed by slight desquamation. Shortly after the subsidence of the rash the fever and pains again return, and persist for two or three days, when convalescence begins.

Diagnosis.—**Acute Rheumatism.**—This disease runs a more protracted course, and lacks the paroxysmal character and the eruption of dengue.

Prognosis.—Favorable.

Treatment.—There is no specific remedy. A mercurial aperient should be given at the onset. The pains are best relieved by phenacetin, salicylates, and morphin. The diet should be liquid and sustaining.

HYDROPHOBIA.

(Rabies.)

Definition.—A specific infectious disease of certain carnivorous animals, especially dogs and wolves, communicated to man by direct inoculation, and characterized by slight fever, intense spasm of the muscles of the throat, delirium, paralysis, and coma.

Ętiology.—Rabies invariably results from the bite of a rabid animal, generally a dog. In the animal the disease is characterized by depression of spirits, loss of appetite, followed by excitement, aimless roving, a morbid desire to bite, and finally by paralysis and death from exhaustion. The poison is contained in the central nervous system and secretions, especially the saliva. Bites on the face and on exposed parts are particularly liable to be followed by infection.

Pathology.—The bacteriology is obscure. Microscopically, the intervertebral ganglia present advanced proliferation of the capsular cells, with degeneration of the ganglion cells, and the medulla and pons, accumulations of deeply staining nuclei around the blood-vessels (vascular tubercles of Babes).

Period of Incubation.—From two weeks to two months.

Symptoms.—The *onset* is characterized by slight fever, anxiety, depression, restlessness, and pain in the wound or cicatrix. In about a day symptoms of the *convulsive stage* appear. These consist in great difficulty in swallowing, severe clonic spasms of the laryngeal muscles, salivation, extreme hyperesthesia, hallucinatory delirium, and prostration. Anything that excites the swallowing reflex, such as the sight of water, may bring on the painful spasm of the throat muscles. In the course of one or two days, if the patient does not die from exhaustion or heart-failure, the *paralytic stage* supervenes, in which the convulsions and delirium give way to ascending paralysis and unconsciousness.

Diagnosis.—**Hysteria** in persons who have been bitten may simulate hydrophobia. Such persons often bark, try to bite, and manifest other symptoms which are not noted in hydrophobia.

Prognosis.—Once developed, the disease is invariably fatal.

Treatment.—*Prophylaxis.*—Suspicious bites should be thoroughly disinfected and cauterized with caustic potash or strong carbolic acid.

The results obtained at the Pasteur Institute at Paris seem to justify the inoculative treatment, in which the person who has been bitten is promptly subjected to a series of inoculations with properly prepared spinal cords from artificially infected rabbits. The treatment of the attack is purely palliative. An attempt should be made to maintain nutrition by rectal alimentation and to control the convulsive paroxysms by injections of morphin and inhalations of chloroform.

CONSTITUTIONAL DISEASES.

RHEUMATIC FEVER.

(Acute Articular Rheumatism; Inflammatory Rheumatism.)

Definition.—An acute disease, characterized by polyarthritis, irregular fever, acid sweats, and a marked tendency to endocardial inflammation.

Etiology.—The disease is most common in the second and third decades. Males are more often attacked than females. It is most prevalent in moist, cold climates. Heredity, lowered vitality, and exposure are predisposing factors. Disposition to recurrence is a characteristic feature.

The exciting cause is still unknown. Most authorities, however, concede that the disease is infectious.

Pathology.—The ligaments and the synovial membrane and its fringes are congested and swollen. The synovial sac is filled with a turbid fluid. The cartilages are roughened and occasionally ulcerated. Generally the process ends in resolution; sometimes the surrounding tissues become infiltrated with inflammatory lymph and false ankylosis results; very rarely, suppuration of the joint follows. The blood shows an excess of fibrin, a considerable diminution in the number of red cells, and an increase in the number of leukocytes.

Secondary inflammations are frequently discovered, such as endocarditis, pericarditis, and pleurisy.

Symptoms.—The symptoms vary much in their severity. The disease usually begins abruptly, or more rarely follows such prodromes as malaise, chilliness, and sore throat. The large joints, especially the symmetric ones, are usually affected; they are slightly reddened, swollen, exquisitely painful, and tender to the touch. The inflammation shows a marked tendency not only to spread from joint to joint, but to disappear abruptly in one while it attacks another. The joints most commonly involved are the knees, elbows, ankles, and wrists, but no joint is exempt. In severe cases the muscles are painful, tender, and somewhat rigid. The fever rises rapidly to a moderate height (102°–103° F.); it is indefinite in its duration and extremely irregular in its course. Perspiration is often copious, has a peculiar sour smell, and an acid reaction. The urine is scanty, high-colored, and on standing throws down an abundant sediment of urates and uric acid. The tongue is heavily coated; the appetite is lost; the bowels are constipated. The face is at first flushed, but as the disease advances it becomes decidedly pale from anemia.

The duration is indefinite, varying from a few days to several weeks.

Complications.—*Vegetative endocarditis* is the most common complication. It occurs in about 40 per cent. of all cases. *Pericarditis* is also common, but less frequent than endocarditis. *Tonsillitis* may occur at the onset of the disease. Certain cutaneous affections—*urticaria*, *purpura*, *erythema nodosum*, and *subcutaneous fibrous nodules* are occasionally met with. *Chorea* sometimes occurs after the acute symptoms have subsided. In exceptional cases the subsidence of the joint inflammation is marked by the development of hyperpyrexia (106°–110° F.), and with it marked cerebral symptoms—delirium, convulsions, coma. To this complication the term *cerebral rheumatism* has been applied.

Rarer complications are pleurisy, iritis, meningitis, and pneumonia.

Diagnosis.—**Septic Arthritis.**—This may be recognized by its association with some other septic process and by the special tendency of the inflammation to end in suppuration, which is a very rare termination of rheumatic arthritis.

Gonorrheal rheumatism may be recognized by the history of gonorrheal infection; its tendency to involve a single joint and to remain in the joint primarily affected; its long duration, and its resistance to salicylates.

Rheumatoid Arthritis.—This begins in the small joints, attacking one after another; leads to permanent deformity; is not associated with fever and acid sweats; and shows no tendency to involve the heart.

Gout.—This occurs later in life, usually involves the great toe, and lacks high fever, acid sweats, and the tendency to heart complications.

Prognosis.—Most cases end in recovery. A very small number die of exhaustion or some complication, such as endocarditis or hyperpyrexia, with grave nervous symptoms. The disease is very prone to relapse and to recur.

Treatment.—Absolute rest in a comfortable bed is essential, and, with the view of preventing permanent injury to the heart, this should be maintained for at least ten days or two weeks after the temperature has become normal and all the arthritic symptoms have subsided. The patient should wear a loose flannel night-dress and lie between blankets. Milk and cereals are the most suitable articles of diet. The free use of water and of lemonade should be encouraged. Two remedies have considerable power in controlling the symptoms: salicylic compounds and the alkaline salts of potassium. From 10 to 15 grains of ammonium or sodium salicylate should be given every two or three hours until a decided impression is made upon the disease or the phenomena of salicylism are produced, when the interval between the doses should be lengthened to four or six hours. It is advisable to continue the drug for several days after the subsidence of the symptoms. When the ammonium or sodium salt is not well borne, strontium salicylate or salophen (1 dram daily) may be substituted.

If the alkaline treatment is employed, 20 to 30 grains of potassium acetate or citrate should be given every two or three hours until the urine becomes distinctly alkaline. It is often a good plan to combine alkalis with salicylates, thus:

℞.	Potassii citratis	ʒiv
	Sodii salicylatis	ʒiij
	Glycerini	f℥j
	Aquæ menthæ piperitæ	q. s. f℥iv.—M.

SIG.—A dessertspoonful every three hours.

Opium, in the form of Dover's powder or of morphin hypodermically, is sometimes of great value in allaying pain, subduing restlessness, and procuring sleep. Antipyrin or phenacetin, in moderate doses, is also a useful adjuvant to salicylates or alkalis when the pain is severe. When adynamia is marked, quinin (5 grains) is frequently beneficial. Anemic patients are benefited by iron.

Hyperpyrexia is best controlled by the cold bath. Endocarditis and pericarditis rarely require special remedies. The importance of prolonged rest in cases in which the heart becomes affected cannot be overestimated. During convalescence tonics, like iron, quinin, and arsenic, and a liberal diet are necessary.

Local Treatment.—In mild cases the joints may be painted with iodin and wrapped in cotton-wool. In severe cases small blisters are of great utility.

Among other effective remedies may be mentioned methyl salicylates or oil of gaultheria (undiluted on compresses); guaiacol (with equal parts of glycerin); lead-water and laudanum (ice-cold or hot), and chloroform liniment. An ointment of salicylic acid is often very useful:

℞.	Acidi salicylici	ʒiss
	Olei terebinthinæ	fʒj
	Adipis benzoinati	q. s. ℥ij.

SIG.—Spread on lint and keep in place by means of a flannel binder.

No matter what local remedy is selected, it is highly important that the affected joints should be kept at complete rest. This may be accomplished by means of padded splints and a roller bandage.

Lingering swelling will often yield to an ointment of mercury and belladonna, with firm strapping of the articulation. Blisters are also useful. When the effusion is very great and persistent, it may be necessary to aspirate the joint.

For the stiffness of the joints massage, warm baths, and inunctions with an ointment of iodin will be found useful. The hot-air treatment also does good in some cases.

CHRONIC ARTICULAR RHEUMATISM.

Etiology.—Chronic articular rheumatism nearly always begins as a chronic affection. Heredity, advanced years, and habitual exposure to cold and wet are predisposing factors. It rarely results from an acute attack.

Pathology.—The fibrous structures around the joint are greatly thickened, so that in long-standing cases the movements are restricted; the neighboring muscles are wasted from disuse; and the nerves often reveal evidences of neuritis.

Symptoms.—Pain, stiffness, deformity, and creaking of the joints are the usual phenomena. Several joints are commonly affected, and the disease shows no predilection for any particular joint. The symptoms grow worse on the approach of stormy weather, and at such times exacerbations are liable to occur, in which the joints become swollen and tender. The duration is indefinite.

Prognosis.—Generally unfavorable. Much relief may follow persistent and judicious treatment, but perfect cure is rarely attainable.

Treatment.—Especial attention should be given to the hygiene, particularly as regards diet, bathing, clothing, exercise, and occupation. A change of residence to a dry, warm, and equable climate is always desirable.

Hot sulphur and hot saline baths are often very useful. Tonics and certain alteratives, such as cod-liver oil, iron, arsenic, guaiac, and potassium iodid are the most generally useful internal remedies:

℞. Liquoris potassii arsenitis . . . . fʒj–ij
Potassii iodidi . . . . ʒij–iij
Syrupi sarsaparillæ compositi . q. s. f℥iij.—M.
SIG.—A teaspoonful in water three times a day after meals.

Local Treatment.—Massage, if employed systematically, often accomplishes much good. Superheated air-baths are occasionally useful. Electricity is of little value. Rubefacient liniments have a palliative influence in mild cases. An ointment of mercury, belladonna, and ichthyol, well rubbed into the affected part, is sometimes very efficacious. When the pain is severe and persistent, blisters or light applications of the actual cautery prove effective.

OTHER MANIFESTATIONS OF RHEUMATISM.

Muscular Rheumatism (Myalgia; Myodynia).—An affection of the voluntary muscles, characterized by pain, tenderness, and rigidity.

Types.—Different names have been applied according to the location, namely: *Torticollis*, or *wry-neck*, when it involves the sternocleidomastoid muscles; *lumbago*, when it involves the lumbar muscles; *pleurodynia*, when it involves the intercostals; and *cephalodynia*, when it involves the occipitofrontalis.

Etiology.—The gouty or rheumatic diathesis is a predisposing cause. Exposure to cold and wet or muscular strain usually excites it.

Symptoms.—Pain is the chief symptom; it is made worse by use of the muscles, and is associated with tenderness which is especially marked at the tendinous origins and insertions of the muscles. Sometimes the muscles are contracted and rigid; this is particularly the case in torticollis, or wry-neck.

Torticollis.—The head is fixed and inclined to one side; every effort to turn it is attended with sharp pain.

Lumbago.—There is a dull, aching pain across the loins. Turning the body or rising from the sitting posture causes an exacerbation, which is sometimes so severe that the patient cries out. Care must be taken to distinguish it from renal calculus, Pott's disease, aneurysm, perinephritis, and uterine or ovarian disease.

Pleurodynia.—The pain is felt in the side, and is increased by deep breathing, coughing, or twisting the body. There is diffuse tenderness to the touch. The absence of fever and of physical signs will serve to distinguish it from *pleurisy*.

The absence of tender spots where the nerves make their exit from the muscular coverings, the fact that the pain does not follow closely the distribution of the nerves, and that the pain is increased by movement, will serve to distinguish pleurodynia from *intercostal neuralgia.*

Cephalodynia.—This is characterized by a superficial pain in the head, increased by moving the scalp. It is often associated with tenderness on pressure.

Prognosis.—Favorable under judicious and persistent treatment.

Treatment.—In mild cases it will suffice to put the affected muscles at rest. In pleurodynia this is accomplished best by strapping the affected side as in fracture of the ribs, and in lumbago by applying a large piece of adhesive plaster from the floating ribs to the iliac crests. In more severe cases it will be necessary to apply rubefacient liniments, sinapisms, or, better still, hot fomentations, and to administer a salicylate, combined, perhaps, with phenacetin:

℞. Salophen . . . . ʒij
Acetphenetidini . . . . ʒj.—M.
Fiant chartulæ No. xij.
Sig.—One every three hours.

A blister is occasionally required. When the pain is intense, intramuscular injections of morphin ($\frac{1}{4}$ grain) with atropin ($\frac{1}{120}$ grain) will afford great relief. In lumbago, acupuncture sometimes yields excellent results. Hot packs and baths are often efficacious, but great care must be exercised to guard against exposure after their use. Persistent myalgia is often very favorably affected by massage and applications of the faradic current. In chronic cases potassium iodid and guaiac should be tried. Gelsemium in large doses (Brunton) and ammonium chlorid (Ringer, Roberts, DaCosta) have also been recommended.

Neural Manifestation.—Rheumatism appears to be a frequent cause of neuritis.

Rheumatic Affections of Mucous Membranes.—It must be borne in mind that pharyngitis, tonsillitis, laryngitis, and bronchitis are sometimes dependent upon a rheumatic diathesis.

Rheumatic Affections of Serous Membranes.—Endocarditis, pericarditis, pleuritis, iritis, and meningitis may be excited by rheumatism.

Cutaneous Manifestations.—Purpura, urticaria, and erythema nodosum are sometimes associated with rheumatism.

GOUT.

(Podagra.)

Definition.—A disturbance of metabolism, characterized in its typical form by deposits of sodium biurate in the joints and other structures, and by recurrent attacks of arthritis.

Etiology.—Gout most frequently develops in the third and fourth decades. It is more common in males than in females. It is often hereditary. The excessive use of wines or malt liquors, overeating, sedentary habits, nervous strain, and chronic lead-poisoning are predisposing factors.

Pathology.—The pathology of gout is still obscure. It is generally conceded that the disease is in some way associated with an excess of uric acid compounds in the blood; but whether these compounds are the sole cause of the constitutional disturbances, and whether the excess in the blood is due to increased formation or diminished excretion, or both, are questions that await solution.

The only distinctive anatomic lesions of gout are those of the joints. These consist of deposits of sodium biurate (tophi) in the cartilages and fibrous tissues and secondary inflammatory changes. In long-continued cases the joints become irregularly enlarged and stiff. Ultimately ulceration of the superficial tissues may ensue, with the discharge of the uratic concretion. The small joints of the feet and hands are usually the first to be affected, but subsequently other joints, like those of the ankles, wrists, and elbows, become involved. Uratic deposits are often found also along the tendons, in the external ear, in the nose, and in various other parts.

In *acute* cases the affected joint, most frequently the metatarsophalangeal of the great toe, is intensely hyperemic, swollen, and edematous.

Chronic interstitial nephritis, arteriosclerosis, and hypertrophy of the heart are important concomitant lesions.

Clinical Varieties.—(1) Articular gout, which may be acute or chronic; (2) non-articular or irregular gout.

Symptoms.—Acute Gout.—The attack is usually preceded by certain prodromes—restlessness, insomnia, moroseness, irritability, dyspepsia, and changes in the urine, this secretion being scanty, high-colored, and deficient in urates. The arthritic phenomena usually appear suddenly in the early morning hours, and are characterized by pain and swelling in the ball of the great toe. The affected joint is so tender that the slightest pressure causes agony. It is of a reddish-purple color; its surface is glazed; and the neighboring veins are full and distinct. During the paroxysm the temperature is moderately elevated (101°–102° F.) and the pulse quickened. Toward daylight the pain subsides to a great extent and the patient falls asleep. During the day he is comparatively comfortable, but there are severe exacerbations for several successive nights. At first the attacks may be a year apart, but as they multiply the interval grows less, until finally the patient is seldom entirely free from suffering.

Retrocedent Gout.—This term is applied to a condition in which the arthritic phenomena suddenly subside and grave gastric, cardiac, or cerebral symptoms follow.

Chronic Gout.—The joints are affected one by one, and become stiff, irregularly enlarged, and deformed. Chalk-stones, or tophi, sometimes ulcerate their way through the skin and are discharged. Similar deposits are frequently found along the tendons and in the helix of the ear.

Constitutional symptoms similar to those occurring in non-articular or irregular gout are more or less conspicuous.

Non-articular Gout (Uric Acid Diathesis; Latent Gout; Goutiness; Lithemia).—This form of gout is more often met with in America than the articular variety. It presents the following clinical features:

Gastro-intestinal Phenomena.—The tongue is generally coated and the breath heavy; the appetite is variable: sometimes it is lost, at others it is inordinate; acid eructations, heart-burn, and flatulence are frequent gastric symptoms.

Urinary Phenomena.—The urine is scanty, high-colored, of high specific gravity (1025 to 1035), and on standing throws down an abundant brick-dust sediment. The solids render the urine irritating, so that dull aching in the loins and burning in the penis after micturition are common symptoms. A trace of sugar is sometimes detected on chemical examination. The urine often stains the clothes red.

Circulatory Phenomena.—These consist in increased arterial tension, accentuation of the second aortic sound, and a tendency to arteriosclerosis.

Nervous Phenomena.—These are extremely varied, and include headache, vertigo, disturbed sleep, tinnitus aurium, depression of spirits, failure of memory, loss of energy, irritability, and neuralgic pain in various parts of the body.

Complications and Sequelæ.—These include: Chronic interstitial nephritis, arteriosclerosis, hypertrophy of the heart, angina pectoris, apoplexy, chronic bronchitis, and certain cutaneous affections—chronic eczema, urticaria, and psoriasis.

Diagnosis.—**Acute Rheumatism.**—This more commonly affects the larger joints; it is markedly migratory; it is associated with higher fever and more copious perspiration; and it shows far greater tendency to endocardial and pericardial inflammations.

Rheumatoid Arthritis.—This occurs more frequently in women than in men; it is more likely to begin in the fingers than in the toes; it usually involves symmetric joints. It is more apt to involve the spinal and temporomaxillary joints; it causes more deformity and fixation of the joints, and, finally, it is not associated with tophaceous deposits in the joints or other tissues, nor necessarily with arterial or renal complications.

Prognosis.—*Acute gout* rarely proves fatal; recurrence, however, is to be expected. On account of the tendency to arterial and renal complications, the prognosis of chronic gout, when the disease is fairly established, should be somewhat guarded. It is largely proportionate to the mildness by the symptoms and the extent to which the patient can be controlled.

Treatment.—*The Acute Attack.*—The best remedy is colchicum: 10 to 20 drops of the wine well diluted should be given every two hours, and stopped as soon as the symptoms subside. Alkalis are valuable adjuncts. The free use of water should be encouraged. Constipation should be relieved by a full dose of blue-mass or a saline draft. Opium or phenacetin may be required for the relief of the pain. The affected part should be elevated and wrapped in cotton-wool, or covered with warm fomentations or with cloths soaked in lead-water and laudanum. The diet should be light and non-stimulating.

Chronic Gout.—As regards diet, simplicity and moderation are of the utmost importance. Generally speaking, a diet composed for the most part of milk, farinaceous foods, succulent vegetables, eggs, fish, is most suitable. The foods most likely to disagree are veal, liver, sweetbreads, hashes, croquettes, concentrated soups, vegetables rich in nucleins,—peas and beans,—pastry, sweets, coffee, malt liquors, and heavy wines. Some patients are exceedingly intolerant of acid fruit.

Water-drinking between meals should be encouraged. No more should be eaten than is absolutely necessary to satisfy hunger. The patient should be warmly clothed and should avoid as far as possible exposure to sudden atmospheric changes. Systematic exercise in the open air is extremely beneficial. When active exercise is not feasible, massage may be strongly recommended. All overwork of mind should be forbidden. Hydrotherapy—tepid sponge-baths and douches—is useful. Heavy, robust patients often derive much benefit from the Turkish bath. Visits to certain minerals springs—Bedford, Saratoga, Harrowgate, Carlsbad, Contrexeville, Aix-les-Bains—are sometimes of great value.

Free action of the bowels should be secured. The occasional use of calomel or blue-mass at night, with a saline in the morning, is often of value. Among the special remedies advocated for gout may be mentioned alkalis and alkaline mineral waters, colchicum, guaiac, arsenic, and iodids.

Of these, the alkalis, especially the vegetable salts of potassium or lithium, are the most useful. Colchicum is most effective in the acute paroxysms, although small doses with alkalis may be of benefit in the interval. Guaiac probably ranks next in efficacy to the alkalis. The prolonged use of arsenic in small doses seems to be of some value. Iodids are sometimes of service in relieving the concomitants and sequels of gout, but have little, if any, effect upon the disease itself. Salicylates relieve pain, but are distinctly inferior to colchicum.

Chronic affections of the joints are best treated by gentle massage, friction, and warm sulphur baths.

RHEUMATOID ARTHRITIS.

(**Arthritis Deformans; Rheumatic Gout.**)

Definition.—A chronic affection of the joints, characterized by destruction of the cartilages, new osseous formations, immobility, and deformity.

Etiology.—It develops most frequently in the third and fourth decades. Women are much more often attacked than men. Heredity, prolonged mental strain, and enfeeblement of health from bad hygienic environment, poor food, or prolonged lactation are predisposing factors.

Pathology.—The origin of rheumatoid arthritis is obscure. Some regard it as a trophoneurosis, allied to the arthropathies met with in certain diseases of the spinal cord. Others believe it to be infectious.

The cells of the cartilages and of the synovial membrane proliferate and lead to villous or nodular outgrowth, which may subsequently be transformed into osteophytes. The central portions of the cartilages ultimately wear away and leave the bones exposed. The heads of the bones become smooth, hard, and shiny. The periarticular tissues are also thickened. The deformity leads to stiffness and ankylosis. Subluxations are common. The surrounding muscles are generally atrophied. All joints are liable to be affected.

Symptoms.—It may be either acute or chronic, the latter being the more common form. In the *acute form* several joints are simultaneously involved; they become swollen, painful, and tender, but rarely reddened. There is moderate fever. The symptoms soon subside, to reappear, however, at frequent intervals.

In the *chronic form* the hands, particularly the metacarpophalangeal joints, are usually first affected; then the wrists, knees, toes, jaws, and spine. Symmetric joints are usually attacked. The symptoms are swelling, pain, immobility, and deformity. The joints are stiff and creak when moved; later complete ankylosis develops. The muscles waste, and contractures increase the deformity.

In advanced cases the fingers are bent backward, often locked, and turned toward the ulnar side; the thighs are drawn up; the legs are adducted and flexed. The patient may be a helpless invalid for many years.

Heberden's Nodes.—These are small nodules that develop gradually on the sides of the distal phalanges, especially of the fingers. At times they may be slightly painful and tender. They are usually seen in middle-aged women. They are usually regarded as an expression of rheumatoid arthritis.

Diagnosis.—The differential diagnosis between rheumatoid arthritis and gout has already been considered (see p. 348).

Chronic Rheumatism.—This usually involves fewer joints; it attacks especially the larger joints; it very rarely involves the spinal or temporomaxillary joints; it does not cause enlargement of the ends of the bones; it shows greater tendency to involve the heart; and it is less steadily progressive.

Prognosis.—Unfavorable. Sometimes the disease is local and remains in one joint (monoarticular form). Generally, however, several joints are affected, and while judicious and persistent treatment may retard the progress of the disease, a cure is rarely attainable.

Treatment.—Hygienic treatment is most important. Tonics, especially iron, arsenic, and cod-liver oil, are generally required. Salicylates are sometimes of service in acute exacerbations. Massage is valuable in preserving the mobility of the joints and in maintaining the nutrition of the muscles.

RICKETS.

(Rachitis.)

Definition.—A constitutional disease of early childhood, characterized chiefly by defective nutrition of the osseous structures.

Etiology.—Rickets is rarely congenital; it usually develops between the first and second years. Poverty, artificial feeding, and bad hygienic conditions are the predisposing causes.

Pathology.—The most marked changes are observed in the long bones and ribs. The cartilaginous lamina between the epiphysis and the shaft are considerably thickened, and are spongy and irregular in outline; microscopic examination reveals an excessive proliferation of the cartilage cells, with scanty calcification. The periosteum is thickened and highly vascular, and when stripped off, soft, porous bone is exposed. The bones are soft, being extremely deficient in lime-salts; when ossification finally results, the bones become heavy, large, and irregular in outline; these changes correspond to the clinical phenomena—bow-legs, knock-knees, spinal curvature, pigeon-breast, and square cranium.

The liver and spleen are often considerably enlarged.

Symptoms.—The early symptoms are: Restlessness and slight fever at night; free perspiration about the head; diffuse soreness and tenderness of the body; pallor; slight diarrhea; enlargement of the liver and spleen; delayed dentition, and the eruption of badly formed teeth.

Skeletal Phenomena.—The head is large and more or less square in outline; careful palpation may detect soft areas. The sides of the thorax are flattened; the sternum is prominent; nodules can be felt at the sternal ends of the ribs—"rachitic rosary"; there may be a distinct transverse groove at the level of the ensiform cartilage; the spinal column is frequently curved anteroposteriorly or laterally; the long bones are curved and prominent at their extremities.

Complications.—These include: Green-stick fractures, convulsions, laryngismus stridulus, paresis of the extremities, and acute pulmonary disease.

Treatment.—The general nutrition must be improved by placing the child under the best hygienic conditions. When hand-feeding becomes necessary, fresh cow's milk, properly modified to suit the age of the infant, egg-albumen, and fresh meat-juice should be recommended. Cod-liver oil is a valuable nutrient tonic. Syrup of the iodid of iron (3 to 20 drops thrice daily) is indicated when there is anemia.

℞. Olei morrhuæ . . . . f℥iss
Olei sassafras
Pulveris acaciæ
Pulveris sacchari . . . . . āā q. s.
Syrupi ferri iodidi . . . . fʒij–iv
Aquæ . . . . q. s. ad f℥iij.—M.
Sig.—A teaspoonful to a dessertspoonful after meals.

Phosphorus (5 to 15 minims of the official elixir thrice daily) is regarded as being especially efficacious by many authorities. It may be added to the cod-liver oil.

DIABETES.

(Diabetes Mellitus.)

Definition.—A nutritional disease, characterized by the persistent presence of sugar in the urine, polyuria, and loss of flesh and strength.

Etiology.—The disease occurs most frequently between the ages of thirty and sixty. It is much more common in males than in females. Hebrews appear to be especially prone to it. Heredity, overeating, sedentary habits, and prolonged mental anxiety are predisposing factors.

Pathology.—The condition which is really responsible for diabetes is still undetermined. Puncture of the floor of the fourth ventricle will produce glycosuria, but the cases are rare in which lesions of this region have been found after death.

In a large number of cases macroscopic or microscopic lesions are found in the pancreas. It has been shown, however, by Opie and others, that diabetes is absent in pancreatic disease unless the lesions are such as destroy the islands of Langerhans. Cirrhotic and degenerative changes are frequently found in the liver. The kidneys are commonly the seat of hyperemia and catarrhal inflammation.

According to Lepine and others, the disease is due to the accumulation of glucose in the blood, owing to the absence of a sugar-splitting ferment (glycolytic ferment) which the pancreas normally manufactures. In the present state of our knowledge, however, pancreatic disease cannot be assumed in all cases. It may be that the hyperglycemia in some instances results from a failure of the liver (owing to actual disease or to functional disturbances induced by influences emanating from the central nervous system) to store up or to retain the carbohydrates.

Symptoms.—*Urinary Phenomena.*—The urine is increased in quantity, the amount varying from three or four pints to as many quarts; its color is pale; its specific gravity usually ranges from 1030 to 1050; it has a sweetish taste and an aromatic odor. In summer it attracts flies and rapidly ferments. It may leave a whitish residue on the clothes. The percentage of glucose varies from 0.5 per cent. to 10 per cent.

General Phenomena.—There is loss of flesh and strength; the temperature is normal or subnormal; the appetite is often inordinate, and the thirst unquenchable; the tongue is often fissured and beefy red; the bowels are usually constipated. The muscles are sometimes the seat of painful cramps.

Cutaneous Phenomena.—The skin is harsh and dry, and frequently the seat of intense itching. Pruritus is especially observed at the genitalia, and this may be the first subjective symptom.

Nervous Phenomena.—These are: Headache, depression of spirits, diminished or lost patellar reflexes, impaired sexual power, dimness of vision, and neuralgia.

The duration varies from a few weeks in the acute form to many years in the chronic form.

Complications.—These include: Pulmonary tuberculosis; pneumonia; gangrene of the lung; defective vision from soft cataract, retinitis, or atrophy of the optic nerve; cutaneous lesions, as boils, eczema, carbuncles, and gangrene; nephritis; neuritis, and diabetic coma, or *acetonemia.*

This last condition is characterized by epigastric pain, dyspnea, a fruity odor of the breath, headache, delirium, stupor, and coma. It is believed to be due to the presence in the blood of β-oxybutyric acid.

Diagnosis.—Care must be taken to distinguish *simple glycosuria* from diabetes. The former is recognized by being transient, and unassociated with the constitutional symptoms of diabetes.

Pruritus and apparently causeless loss of flesh and strength should lead to a suspicion of diabetes.

Prognosis.—The younger the patient, the stronger the hereditary tendency, the larger the amount of sugar excreted, the less the glycosuria can be controlled by diet alone, the graver the prognosis. On the other hand, when it occurs after middle life in association with a gouty diathesis, and the glycosuria is not pronounced, the prognosis for a long duration is comparatively favorable. Absolute cure is rarely attainable.

Treatment.—*Dietetic Treatment.*—Sugars and starches must be restricted. Since the patient's appetite is often inordinate, it is necessary also to regulate the quantity and character of the foods that are recognized as admissible. The following foods may be included in the dietary:

Animal Foods.—Meats of various kinds (except liver), game, light broths and soups, fish, and eggs.

Vegetables.—Celery, lettuce, cauliflower, tomatoes, mushrooms, string-beans, young onions, olives, water-cress, and spinach.

Beverages.—Buttermilk, skim milk, sour wines (Rhine wines), carbonated waters, and coffee and tea without sugar.

Relishes.—Nuts of all kinds (except chestnuts), cream cheese, and pickles.

Bread.—Bread made of gluten, bran flour, or almond flour. It should be borne in mind that all the gluten flours are rich in starch.

Fruits.—Cranberries, sour cherries, limes, lemons, and red currants.

Substitutes for Sugar.—Saccharin and glycerin.

The following foods should be avoided: Liver, oysters, wheat bread, biscuits, pastry, potatoes, beets, carrots, peas, turnips, parsnips, sweet fruits, rice, barley, tapioca, cornstarch, corn-meal, chocolate, cocoa, syrups, preserves, and most liquors.

Hygienic Treatment.—Fresh air and systematic exercise are of great value. The patient must be warned, however, against overexertion. Flannel should be worn next to the skin, and all undue exposure avoided. Hydrotherapy is decidedly efficacious. Diabetics who still possess a fair measure of health frequently derive much benefit from a visit to certain mineral springs, such as Neuenahr, Homburg, Carlsbad, and Vichy.

Medicinal Treatment.—Tonics, like arsenic, strychnin, and cod-liver oil, are often indicated. Opium is generally the most reliable special remedy; it should be given in small doses gradually increased until the patient takes 5 or 6 grains daily. Codein (½ grain increased to 6 grains a day) is sometimes preferable to opium. Salicylic compounds rank next in efficacy to opium. From 40 to 60 grains of ammonium or strontium salicylate may be given in twenty-four hours.

℞. Strontii salicylatis	ʒiv–vj
Liquoris potassii arsenitis	f℥j
Glycerini	f℥j
Aquæ cinnamomi . . . . . . q. s. ad	f℥iv.—M.

SIG.—Dessertspoonful thrice daily.

Alkaline carbonates and alkaline mineral waters have long enjoyed a reputation. Bromids are useful in subduing nervous symptoms. Among remedies that occasionally succeed may be mentioned Clemens's solution of arsenic bromid (3 to 5 minims); antipyrin (8 to 10 grains thrice daily); and jambul.

Diabetic coma is always fatal, but inhalations of oxygen or the subcutaneous injection of large quantities of normal saline solution at intervals may give a few hours' respite, in which consciousness returns.

DIABETES INSIPIDUS.

Definition.—A chronic condition, characterized by the excretion of large quantities of pale, limpid urine of low specific gravity and free from albumin and sugar.

The condition must be distinguished from the polyuria observed in chronic interstitial nephritis and in some cases of hysteria.

Etiology.—It is most common between the ages of twenty and thirty. More males are affected than females. Heredity is an important etiologic factor. It is sometimes associated with lesions in the neighborhood of the medulla or floor of the fourth ventricle, such as tumors, hemorrhages, and especially syphilitic basilar meningitis. In a few cases it appears to have followed intense emotional excitement.

Pathology.—Little is known of the pathology. The kidneys are frequently enlarged and congested, and the ureters dilated.

The theory which is generally accepted as accounting for the polyuria is that it is due to a vasomotor paresis of the renal vessels, which permits a free transudation of liquid.

Symptoms.—The disease may begin insidiously or abruptly; the latter is the rule. *The urine:* The quantity is increased, often as much as eight or ten quarts being excreted in the twenty-four hours; it is pale, and resembles water; it has a specific gravity of 1002 to 1005. The total amount of solids is not diminished. Albumin and sugar are generally absent, though there may be a trace of the latter.

The most important *general symptoms* are extreme thirst; dryness of the skin; constipation; mental apathy; and exaggeration of the knee-jerks. In many cases there is neither weakness nor emaciation. Complications are rare.

Diagnosis.—The high specific gravity of the urine and the presence of sugar will serve to distinguish **diabetes mellitus** from diabetes insipidus.

Interstitial nephritis may be recognized by the presence of tube-casts in the urine, the albuminuria, and cardiovascular signs.

Prognosis.—The duration of the disease is very indefinite. Not infrequently spontaneous cure occurs. Unless the result of a serious nervous lesion, it rarely terminates fatally.

Treatment.—No benefit is derived from restricting the quantity of water desired. Acidulated drinks, like lemonade, aid in assuaging thirst. Many remedies have been recommended; those possessing the most extended reputation are opium (4 to 8 grains a day), valerian (½ to 1 fluidounce of ammoniated tincture daily), ergot (½ to 1 dram of the extract daily), antipyrin (10 grains thrice daily), and gallic acid (1 dram a day). Galvanism—one pole applied to the neck and the other to the loins—has been recommended. Tonics—cod-liver oil, iron, and strychnin—are sometimes required. In syphilitic cases good results not infrequently follow the use of antiluetic remedies.

SCURVY.

(Scorbutus.)

Definition.—A disease characterized by marked weakness, anemia, hemorrhages from the mucous membranes and into the skin, and a tendency to a spongy state of the gums.

Etiology.—The chief predisposing causes of scurvy in adults are unhygienic surroundings and a dietary deficient in fresh vegetables.

Symptoms.—These include anemia with great weakness and lassitude; spongy, bleeding gums with fetor of the breath and loosening of the teeth; subcutaneous ecchymoses and hemorrhages from the mucous membranes; and brawny induration of the muscles in various parts of the body from a sanguineous transudation.

An infantile form of scurvy (*Barlow's disease*) sometimes follows the prolonged use of condensed milk, sterilized milk, or proprietary foods. The characteristic symptoms are: Pallor, tenderness or pain in the legs or back on handling, slight swelling, especially about the diaphyses, immobility of the legs (pseudoparalysis), ecchymoses, and hematuria. The gums are usually affected when there are teeth, but very rarely when there are no teeth.

Prognosis.—Favorable in its earlier stages.

Treatment.—The diet should include fresh vegetables—potatoes, lettuce, cabbage, and onions—with several ounces of lemon-juice daily. Iron is of service. The mouth should be cleansed at frequent intervals with some antiseptic wash.

In infantile scurvy good results follow the use of fresh milk, beef-juice, and orange-juice.

HEMOPHILIA.

(**Bleeder's Disease; Hemorrhagic Diathesis.**)

Definition.—A hereditary disease, characterized by a tendency to bleed excessively from slight wounds or even spontaneously.

Etiology.—The chief cause is heredity. It is more common in males, but it is generally transmitted by females, even by those who are not themselves afflicted.

Pathology.—The exact cause of the hemorrhages is unknown. In a few instances the walls of the vessels have been found unnaturally thin and degenerated.

Symptoms.—The chief symptom is free and persistent bleeding after trivial injury. Spontaneous hemorrhages from mucous membranes of the nose, stomach, bowel, etc., and subcutaneous extravasations are also quite common. The only other symptom is a peculiar inflammation of the joints, resembling rheumatism.

Prognosis.—Unfavorable. Grandidier states that one-half die before the eighth year, and less than one-eighth survive their twenty-first. In some instances the tendency is outgrown.

Treatment.—This is chiefly protective and palliative. Bleeding will call for rest, the application of cold compresses and of styptics, and the administration of internal hemostatics. The drugs most worthy of confidence are gelatin, calcium chlorid, and thyroid extract.

PURPURA HAEMORRHAGICA.

(Morbus Maculosus Werlhofii.)

Definition.—A disease arising without obvious cause, and characterized by extravasation of blood into the subcutaneous tissues and bleeding from the mucous membranes.

Etiology.—The cause is unknown. An infectious origin is not unlikely. The disease occurs most frequently in young adults, especially in debilitated girls.

Symptoms.—The onset may be marked by moderate fever (102° to 103° F.), headache, malaise, and pain in the limbs; but these symptoms may be absent, and the disease ushered in with a copious purpuric eruption, followed by bleeding from the mucous membranes. Anemia and its associated phenomena develop in severe cases.

Diagnosis.—**Scurvy** is to be distinguished by a history of dietetic errors; by the spongy state of the gums; and by the brawny induration of the muscles. **Hemophilia** may be recognized by the family history and the tendency to arthritis.

Prognosis.—This depends on the severity. Mild cases recover in from one to two weeks; severe cases may prove fatal in a few days from exhaustion or from hemorrhage into the brain. Relapses are not uncommon.

Treatment.—The patient should be put to bed and placed upon a nourishing diet. Among the many remedies advocated ergot, turpentine, tincture of ferric chlorid, calcium chlorid, and gelatin enjoy the most favor.

DISEASES OF THE NERVOUS SYSTEM.

DISTURBANCES OF MOTION.

THESE consist, for the most part, of loss of power or paralysis, and manifestations of motor excitation, such as convulsions, choreiform movements, and tremors.

Paralysis.—The paralysis may be irregularly distributed, or it may involve a single member, when it is termed *monoplegia;* a lateral half of the body, when it is termed *hemiplegia;* or the body from the waist down, when it is termed *paraplegia.*

Irregular paralysis may result from:

1. Disseminated lesions in the motor areas of the brain, which are commonly syphilitic.

2. Lesions in the basal ganglia,—pons, crura cerebri, medulla,—when it is often associated with headache, vomiting, vertigo, and optic neuritis.

3. Acute poliomyelitis. This develops abruptly; it occurs in young children; and it is followed by rapid improvement in some muscles and permanent atrophy and paralysis in others.

4. Chronic poliomyelitis. This develops in middle life; begins in the small muscles of the hand; is associated with atrophy, and progresses very slowly.

5. Muscular dystrophies. These commonly develop during childhood; involve the muscles of the calves, trunk, face, or shoulder-girdle; are associated with progressive atrophy; and are generally traceable to heredity.

6. Multiple neuritis. This is recognized by the history, pain, disturbances of sensation, and tenderness over the nerve-trunks.

7. Syringomyelia. This is rare, develops during adolescence, and is recognized by lateral spinal curvature, fibrillary tremors, atrophy of the affected muscles, various trophic disturbances, and a loss of thermic and painful sensations, while tactile sensation is retained.

Monoplegia may result from:

1. A focal lesion in the cortical area of the brain. This may be recognized by the history, the absence of wasting, of sensory disturbances, and of the reactions of degeneration.

2. A lesion of the peripheral nerve, from traumatism, neuritis, or the pressure of a tumor. Brachial monoplegia frequently results from the pressure of the head on the arm during sleep. Monoplegia of peripheral origin is recognized by the history, the wasting, the sensory disturbances, and the presence of reactions of degeneration.

3. Hysteria. This may be recognized by the history, sex, and temperament; the paroxysmal character of the paralysis; the disturbances of sensation; and contractures without atrophy or electric disturbances.

Facial monoplegia may result from a small lesion in the facial center of the cortex or in the medulla; or from involvement of the nerve in the canal of the temporal bone; or after its exit from the stylomastoid foramen.

Facial diplegia (double facial paralysis) generally results from a lesion at the base of the brain.

Hemiplegia may result from:

1. A diffuse lesion of the motor cortex. The paralysis is on the opposite side of the body and is unassociated with anesthesia.

2. A lesion of the internal capsule or the adjacent ganglia (corpus striatum and optic thalamus). This is the most common seat of hemorrhage; the paralysis is on the opposite side of the body and is rarely associated with anesthesia.

3. A lesion of the crus cerebri. This frequently produces hemiplegia and hemianesthesia on the opposite side, and paralysis of the oculomotor nerve on the side of the lesion, indicated by dilated pupil, strabismus, and ptosis.

4. A lesion of the pons. This frequently produces hemiplegia and hemianesthesia on the opposite side, and facial paralysis on the side of the lesion.

5. A unilateral lesion high in the cord (very rare). This produces a spastic paralysis on the side affected, and hemianesthesia on the opposite side ("Brown-Séquard's paralysis").

6. Hysteria. This may be recognized by the history, sex, and temperament; by being frequently paroxysmal; by its association with sensory disturbances; by the absence of wasting and of abnormal electric reactions; and by the escape of the facial muscles.

Paraplegia may result from:

1. Hemorrhage into the cord at the dorsal region. The paralysis develops abruptly, and is associated with complete anesthesia and involvement of the bladder and rectum.

2. Hemorrhage into the membranes of the cord. The paralysis develops rapidly, but more slowly than the preceding; is associated with intense tearing pains and incomplete anesthesia.

3. Some forms of multiple neuritis. This may be recognized by the pains, the disturbances of sensation, the tenderness over the nerve-trunks, and the absence of "girdle pain" and sphincter involvement.

4. Fracture of the vertebræ.

5. Acute myelitis. The paralysis develops in the course of a few days, and is associated with anesthesia, bed-sores, involvement of the bladder and rectum, loss of reflexes, and wasting of the muscles.

6. Landry's disease (acute ascending paralysis). This develops in the course of a few days, and is unassociated with anesthesia, wasting of the muscles, bed-sores, or sphincter involvement.

7. Chronic myelitis. This develeps over several years, and is associated with numbness and tingling, increased reflexes, involvement of the bladder and rectum, and anesthesia.

8. Compression of the cord from morbid growths, aneurysm, or spinal caries. This may be recognized by the his-

tory, the symptoms of the primary disease, the anesthesia or hyperesthesia, and the intense pains radiating along the line of the spinal nerves.

9. Lateral sclerosis. This develops slowly and is associated with a spastic condition of the muscles and with increased reflexes, and lacks sensory disturbances.

10. Injury of the brain in delivery (spastic paraplegia of infants). The symptoms resemble lateral sclerosis, and are often associated with imbecility or idiocy.

11. Hysteria. This may be recognized by the history, sex, and temperament; by being frequently paroxysmal; and by the absence of wasting and of abnormal electric reactions.

12. Caisson disease (divers' paralysis). The history will establish the diagnosis.

Convulsions.—A convulsion is a condition in which there are excessive muscular contractions, continued or intermittent, dependent upon an involuntary discharge of motor impulses from the nerve-centers.

Intermittent contractions are termed *clonic;* continued contractions, *tonic.*

Convulsions may be general or local. The term *spasm* is sometimes applied to the latter.

Varieties of Convulsions.—Three varieties are frequently made: (1) Epileptiform; (2) tetanic; (3) hysterical.

Epileptiform Convulsions.—In this form there is unconsciousness, and the movements are for the most part clonic. Epileptiform convulsions may result from:

1. Idiopathic epilepsy. This condition usually develops before the age of twenty-five, and the convulsions are general and are unassociated with any definite cause.

2. Organic brain disease. In this condition there may be a history of syphilis or of injury; the convulsions may be local, or begin as such and become general; and there may be concomitant symptoms of cerebral disease.

3. Toxic agents in the blood. Alcoholism, the infectious fevers, and uremia are frequently associated with convulsions.

4. Reflex irritation. Such convulsions are usually ob-

served in young children, and result from gastric irritation, an adherent prepuce, intestinal parasites, or teething.

5. Cerebral anemia. Such convulsions are sometimes seen after profuse hemorrhage, in fatty heart, and in poisoning from cardiac paralyzants like aconite and veratrum viride.

Eclampsia.—This term is applied to a sudden attack of convulsions, the result of a temporary cause, such as the convulsions of childhood resulting from reflex irritation, and the convulsions of pregnancy resulting from toxic materials retained in the blood.

Tetanic Convulsions.—In this form the discharges emanate from the spinal cord, and are not associated with a loss of consciousness. Tetanic convulsions may result from:

1. Tetanus. This may be recognized by the history of a wound, the tonic character of the convulsions, the early involvement of the jaw, and the absence of fever.

2. Spinal meningitis. This may be recognized by the exquisite pain in the back, Kernig's sign, disturbances of sensation, fever, and late involvement of the jaw.

3. Strychnin-poisoning. This may be recognized by the history, the intermittent character of the convulsions, the absence of fever, and the escape of the muscles of the jaw until very late.

4. Tetany. In this condition the extremities are chiefly involved; the convulsions are intermittent, and can be produced by pressure on the nerves and arteries of the affected limbs.

Hysteric Convulsions.—These are manifestations of hysteria, and in them consciousness is only partially or apparently lost. They are not preceded by an aura, but sometimes by a sensation as of a ball in the throat—the "globus hystericus"; the eyes are partially closed; consciousness is not actually lost; the face is often expressive of some emotion; the tongue is not bitten; the movements are usually tonic, and if clonic, appear purposive; and the paroxysm is of long duration.

Local Convulsions or Spasms.—*Spasm of the face* may result from—(1) A cortical lesion in the inferior portion of the ascending frontal convolution; (2) from *tic convulsif*—a condition occurring in young children affecting the facial and neighboring muscles, and associated with mimicry, a tendency to repeat vulgar phrases, and various mental disturbances; (3) from habit (habit chorea); and sometimes from (4) *tic douloureux*—neuralgia of the fifth nerve.

Temporary spasms of one arm or one leg are usually manifestations of Jacksonian epilepsy (focal epilepsy), but they sometimes result from hysteria.

Spasm of the hand developing when the member is put to use may result from writers' cramp, Thomsen's disease, or hysteria.

Spasm of the cervical muscles (wry-neck, torticollis) may result from congenital shortening of the sternomastoid, myalgia, hysteria, caries of the vertebræ, or the irritation of enlarged cervical glands.

Spasms of the larynx, esophagus, and diaphragm (hiccup) have already been discussed.

Saltatory Spasm.—This term is employed to designate a condition allied to hysteria, in which a violent spasm seizes the muscles of the leg as soon as the feet touch the ground, and as a result the patient is thrown violently into the air.

Salaam Convulsions.—These consist of violent paroxysmal bobbing movements of the head or trunk, and may be associated with hysteria, chorea, or, rarely, organic brain disease.

Choreiform Movements.—These are coarse, jerky, irregular, involuntary movements which more or less simulate purposive movements. They may result from:

1. Acute chorea (St. Vitus's dance). This disease is seen in children; is usually mild; runs a course of several weeks; and is prone to be followed by endocarditis.

2. Chorea insaniens. A grave disease occurring in adults, especially pregnant women, and characterized by violent movements, delirium, and fever.

3. Huntingdon's chorea (chronic chorea). An affection occurring in adult life, generally hereditary, and characterized by irregular movements, disturbance of speech, and increasing dementia.

4. Organic brain disease. Choreiform movements are

frequently observed in cerebral palsies of children; they may also develop on one side of the body before an attack of apoplexy (prehemiplegic chorea), or in the paralyzed members after the hemorrhage (posthemiplegic chorea).

5. Peripheral irritation. Choreiform movements sometimes develop in pregnancy, and are occasionally noted in stumps after amputation.

6. Habit. Children frequently acquire, through constant repetition or mimicry, choreiform movements which may last indefinitely.

7. Hysteria. The marked rhythmic character of the movements and the history will aid in the recognition of hysteric chorea.

8. Disseminated cerebrospinal sclerosis. This disease usually induces tremors, but not uncommonly the movements are choreiform. The increased reflexes, the nystagmus, the loss of power, the spastic gait, and the impairment of intellect will aid in its recognition.

9. Paramyoclonus multiplex. A very rare disease, of unknown origin, characterized by violent clonic spasms of the muscles of the trunk and upper part of the limbs. The spasms are bilateral and rarely, if ever, involve the forearms, hands, legs, or feet. They occur only at intervals and can usually be brought on by irritation of the skin or tendons, or by excitement.

Athetosis.—This term was employed by Hammond to designate certain movements occurring chiefly in the hands and feet, and characterized by slow twisting, intertwining, separation, and extension of the fingers and toes. Athetosis is frequently observed in the cerebral palsies of children, and it occasionally occurs in adults as a result of lesions in the basal ganglia.

Tremors.—A tremor is a fine vibratory movement due to the alternate contraction and relaxation of antagonistic muscles. Tremors are observed in the following conditions:

1. They may exist from birth unassociated with other symptoms.

2. They may depend upon a lowered tone of the nervous system, being frequently observed in exophthalmic goitre, neurasthenia, and in the convalescence from acute disease.

3. They may be toxic, resulting from alcoholism or mercurial poisoning.

4. They may be due to old age.

5. They are frequently a symptom of organic disease of the brain and cord; as such, they are met with in paretic dementia, and especially in disseminated sclerosis.

6. They may be the chief symptom in paralysis agitans.

7. They may be hysterical.

The Gait.—The Ataxic Gait.—In locomotor ataxia the patient raises the foot high, throws it forward, and brings it down suddenly, so that the whole sole comes in contact with the floor at once.

Spastic Gait.—In spastic paraplegia the movements are stiff, the knees come together, the leg drags behind, and the toe catches the ground.

Festination.—This term is applied to the gait of advanced paralysis agitans; in walking, the body inclines more and more forward, and the steps grow faster and faster until the patient falls, straightens himself by a supreme effort, or finds support in some neighboring object.

Steppage Gait.—In chronic multiple neuritis the patient raises the foot high, turns the toe up, and brings the heel down first.

The Gait of Pseudomuscular Hypertrophy.—The feet are wide apart, the belly protrudes, and the movements are clumsy and waddling.

Titubation.—This term is applied to the peculiar gait observed in lesions of the cerebellum. It resembles the gait of locomotor ataxia, but is much more staggering, the body swaying like that of a person intoxicated. With the ataxia there is a marked vertigo, which usually disappears when the patient lies down.

The Reflexes.—The Knee-jerk, or Patellar Tendon Reflex.—This is obtained by tapping the quadriceps tendon between its insertion and the patella while the leg is crossed over its fellow.

The knee-jerk is increased in the following conditions:

1. In brain lesions which abolish the inhibitory influence of the cerebrum, as in hemiplegia from apoplexy, tumor, etc.

2. In compression or partial destruction of the cord above the lumbar region, as in Pott's disease or tumor of the cord.

3. In disseminated cerebrospinal sclerosis, lateral sclerosis, and amyotrophic lateral sclerosis.

4. In irritability of the cord, as in mania, hysteria, strychnin-poisoning, and spinal meningitis.

The knee-jerk is diminished or absent in the following conditions:

1. In the various forms of primary muscular atrophy.

2. In lesions of the nerves which cut off the impulse from the cord—as neuritis.

3. In lesions of the posterior columns of the cord, as in locomotor ataxia.

4. In poliomyelitis, both acute and chronic.

5. In advanced myelitis, when the cord is sufficiently injured.

6. In exhaustion of the spinal centers, as after prolonged muscular exertion.

7. In some cases of complete division of the spinal cord, as by fracture or luxation of the vertebræ.

Ankle-clonus.—This consists of vibratory movements of the foot, obtained by supporting the *tendo Achillis* with one hand while the foot is strongly flexed with the other. It is rarely obtainable in health; it is marked in primary lateral sclerosis and cerebral hemiplegia, and is occasionally present in hysteria.

Arm-jerk.—This is obtained by striking the biceps tendon at the elbow, or the triceps tendon above the olecranon.

Jaw-jerk.—This is obtained by tapping the jaw while the mouth is partially open.

Babinski's Reflex.—This consists in extension of the great toe instead of flexion when the sole of the foot is tickled. It is often normally present in infants. In adults it is suggestive of some disturbance of the pyramidal tracts—meningitis, tumor, hemorrhage, amyotrophic lateral sclerosis, etc.

Kernig's Sign.—This consists in an inability to straighten the leg completely when the patient is in the recumbent posture and the thigh is flexed at a right angle with the pelvis. It is of value in the diagnosis of meningitis.

The Cutaneous or Superficial Reflexes.—These are muscular contractions resulting from irritation of the sensory nerves of the skin. Their nature is imperfectly understood. As they are inconstant in health they are much less serviceable for diagnosis than the deep reflexes. Cutaneous reflexes are frequently lost in those diseases in which the tendon reflexes are exaggerated. The important reflexes of this type are the following:

Abdominal Reflex.—Tickling or shaking the skin of the abdomen causes contraction of the muscles on the side stimulated.

Cremasteric Reflex.—Stroking the inner side of the thigh causes retraction of the testicle.

Plantar Reflex.—Tickling the sole of the foot causes sudden plantar flexion of the toes.

Gluteal Reflex.—Irritation of the skin about the buttock causes contraction of the gluteal muscles.

DISTURBANCES OF SENSATION.

These consist chiefly in a loss of sensation—*anesthesia;* increased sensation—*hyperesthesia;* certain abnormal sensations—*paresthesiæ;* and subjective painful sensations—*neuralgias.*

Anesthesia.—Ordinary cutaneous sensibility may be tested by the prick of a pin, by a pinch, or by the faradic current.

Anesthesia results from interruption of the sensory tract in the nerves, as by neuritis; from interruption of the sensory tract in the cord or brain; from organic disease of the sensory area of the brain; from the action of toxic substances on the nerves or centers; from certain functional conditions like hysteria; and from reflex irritation.

Hemianesthesia.—A loss of sensation in a lateral half of the body. It may result from:

1. Hysteria. This is often unassociated with paralysis of motion, and may be recognized by the history, sex, and temperament of the patient; by the paroxysmal character of the anesthesia; and by exclusion of other causes.

2. A unilateral lesion high in the cord. This is very rare, and may be recognized by being associated with hemiplegia on the opposite side.

3. A lesion of the medulla (very rare). The hemianesthesia is usually associated with hemiplegia, paralysis of the cranial nerves, difficult swallowing, and cardiac and respiratory disturbances.

4. A lesion in the pons. The hemianesthesia is often associated with hemiplegia on the same side and facial palsy on the opposite side.

5. A lesion in the crus or peduncle. The hemianesthesia is often associated with hemiplegia on the same side and paralysis of the oculomotor nerve on the opposite side.

6. A lesion of the posterior limb of the internal capsule or of the optic thalamus pressing on the capsule.

7. A lesion of the cortex immediately back of the fissure of Rolando (posterior central convolution).

Monanesthesia.—A loss of sensation in one member. It may result from hysteria, from a focal lesion of the sensory area of the cortex, or from a lesion of the nerves supplying the member.

Paranesthesia.—A loss of sensation in all parts below the waist. It may result from hysteria, organic diseases of the cord, neuritis of the lower extremities, or reflex irritation.

Thermo-anesthesia.—Insensibility to heat or cold occurring as an independent condition. It is sometimes observed in hysteria and syringomyelia.

Analgesia.—Insensibility to pain. It is sometimes observed in hysteria and in certain organic diseases of the spinal cord, especially syringomyelia.

Astereognosis.—A loss of the power of recognizing objects by touch or a failure of tactile memory. It is a symptom of disease in the sensory area of the cortex (middle third of the posterior central convolution and adjacent part of the inferior parietal lobule).

Retardation of Sensations.—This is frequently observed in all forms of anesthesia, but especially in the anesthesia of locomotor ataxia.

The Sense of Space.—The distance at which two points of contact can be recognized as two points. Normally the distance varies in different parts and in different individuals.

On the cheek it is 11–15 millimeters.
On the forehead, 22 millimeters.
On the forearm, 40 millimeters.
On the chest, 45 millimeters.
On the thigh and upper arm, 68 millimeters.
On the leg, 40 millimeters.
On the palm of the hand, 8–12 millimeters.
On the back of the hand, 31 millimeters.

Hyperesthesia is increased insensibility to external impressions.

It is commonly observed in hysteria, especially in connection with the joints, breasts, genitalia, and spine. It is also observed in neurasthenia and in beginning inflammation of the nerves and of the cerebrospinal meninges.

Paresthesia.—This term is used to indicate certain disagreeable subjective phenomena, such as numbness, tingling, itching, creeping, prickling, etc.

Paresthesia is observed in many conditions, as hysteria, spinal sclerosis, neurasthenia, and injury or inflammation of the nerves. It is frequently observed in elderly persons with arteriosclerosis.

Girdle Sensation.—The sense of having a girdle or tight band around the trunk. It is frequently observed in spinal sclerosis.

Neuralgia.—This consists of paroxysms of severe pain radiating along the line of the nerve-trunks. The pain is relieved by pressure, but there are tender spots (*points douloureux*) where the nerve makes its exit from bony canals or muscular coverings.

Lightning-pains.—This term is applied to the sharp lancinating pains observed in locomotor ataxia. They usually occur in the extremities, and may be mistaken for rheumatism.

Causalgia.—This term has been applied by S. Weir Mitchell to an intensely burning sensation generally observed in "glossy skin."

Pressure Sense.—By this sense the amount of pressure exerted on a given part of the body is determined. It may be tested by placing upon the palms or fingers objects of the same bulk but of different weight, the hands being supported upon a table.

Muscular Sense.—This is the sense by which weight, muscular effort, and position are determined. It is often defective in hysteria, locomotor ataxia, and in many forms of paralysis.

DISTURBANCES OF NUTRITION.

These consist in atrophy of the muscles, changes in electromuscular contractility, tissue metamorphoses, and in certain abnormalities of the appendages.

Muscular Atrophy.—Atrophy or wasting of the muscles results from:

1. Inactivity. Cerebral palsies may thus be associated with very gradual wasting.
2. Lesions of the cells in the anterior gray horns of the cord, as in acute and chronic poliomyelitis.
3. Lesions of the nerves, such as neuritis or traumatism.
4. Certain diseases of the muscles themselves, as the muscular dystrophies.

The atrophy that attends chronic affections of the joints probably results from neuritis.

Changes in Electromuscular Contractility.—A normal response of the muscles to both galvanic and faradic current usually occurs in *hysteric paralysis* and in *paralysis of cerebral origin.* An increased response to both currents without qualitative change indicates a state of hypersensitiveness of the spinal centers or peripheral nerves, and may be observed in *very recent cases of neuritic paralysis* and in *tetany.* A diminished response to both currents without qualitative change is observed in the *muscular dystrophies.*

Reaction of Degeneration (DeR).—This consists in a qualitative change in the electric reaction, a reversal of that occurring in normal muscle. It is obtained only with the *galvanic current* when the electrode is placed over the *muscle*,—not its motor nerve or motor point,—and occurs in paralyzed muscles which are in certain stages of degeneration owing to a lesion of the ganglion cells in the anterior gray horns of the cord or of the prolongations of these cells in the peripheral nerves. Thus it is observed in *acute* and *chronic poliomyelitis*, in *acute myelitis*, and in *severe forms of neuritis*. In these diseases the affected muscles fail to respond to the faradic current, but still respond to the galvanic current. The responses, however, instead of being prompt and short, as in health, are sluggish and persistent, and, moreover, are reversed in their sequence. Thus, the anodal (positive pole) closing contraction may equal, or at a later period exceed, the cathodal (negative pole) closing contraction, and the cathodal opening contraction may equal or exceed the anodal opening contraction. These reactions may be expressed as follows:

An ClC equals or is greater than CaClC. CaOC equals or is greater than AnOC.

Arthropathies.—An arthropathy is a degenerative affection of the joints, characterized by marked swelling due to effusion, erosion of the cartilages, relaxation and calcification of the ligaments, and atrophy of the heads of the bones. Arthropathies are observed in certain organic diseases of the spinal cord, more especially in locomotor ataxia and syringomyelia. Some regard the joint phenomena of rheumatoid arthritis as belonging to this class.

Ulceration Resulting from Perverted Nutrition.—*Acute Decubitus.*—This term is applied to ulcers appearing in a few hours or days, on parts subjected to pressure, after the occurrence of a severe cerebral or spinal lesion.

Chronic Decubitus.—This term is applied to the ulcers which ultimately appear on parts subjected to pressure in the course of chronic spinal affections.

Perforating Ulcer of the Foot.—This term is applied to an undermining ulcer of the foot most commonly observed in locomotor ataxia. It frequently penetrates the deep structures and involves the bones.

Symmetric Gangrene (*Raynaud's Disease*).—This is a gangrenous affection involving the fingers, toes, tip of the nose, or ears. It arises spontaneously, and is probably due to a vasomotor spasm.

Trophic Affections of the Skin.—Herpes, scleroderma, vitiligo, chloasma, and the "glossy skin" following injuries of the nerve-trunks are illustrations of this class of trophic phenomena.

Trophic Affections of the Hair and Nails.—After injury of the nerves and in neuritis the nails often become dry, brittle, and cracked. Under similar conditions there may be a loss of hair, an overgrowth of hair, or a change in the color of the hair.

DISTURBANCES OF CONSCIOUSNESS.

Coma.—Coma is a state of prolonged unconsciousness, somewhat resembling sleep, from which the patient cannot be aroused.

Temporary unconsciousness due to anemia of the brain is termed *syncope*. It may be recognized by the extreme pallor, weak pulse, and feeble heart-sounds. Coma may result from:

1. *Traumatism.*—This can be recognized only by the history or the local evidence of injury.

2. *Organic Disease of the Brain.*—The most common cause under this head is apoplexy, which may be recognized by the history, the age, the condition of the arteries, and by evidences of paralysis, such as stertorous breathing, unnatural relaxation or rigidity on one side of the body, conjugate deviation of the eyes, and a higher temperature in one axilla.

3. *Epilepsy.*—The coma of epilepsy is usually of short duration. It may be recognized by the history, by the bloody saliva, by the presence of scars on the tongue from previous attacks, and by the exclusion of other causes.

4. *Thermic Fever* (*Sunstroke*).—The temperature of the day or of the room in which the patient is found, the extremely high body-temperature, and the absence of other causes will usually prevent an error in diagnosis.

5. *Certain Drugs.*—Under this head come *alcoholism* and

opium-poisoning. In *alcoholism* the patient can generally be aroused by shouting in the ear, there is the odor on the breath, and there is an absence of other causes.

In *opium-poisoning* the pupils are small, the respirations are slow, the temperature is normal or subnormal; there may be the odor of laudanum on the breath. The diagnosis will be aided by the exclusion of other causes.

6. *Uremia.*—In this condition there is a urinous odor to the breath; the aortic second sound is accentuated; the urine is scanty and contains albumin; the temperature may be above or below normal; the pupils are usually small and equal, and there is no evidence of other cause.

7. *The Infectious Fevers.*—The history is sufficient to make the diagnosis. Pernicious malarial fever may produce sudden coma, and in this condition the examination of the blood affords conclusive evidence.

8. *Hysteria.*—The history, age, and sex of the patient and the absence of other cause will suggest the condition.

9. *Acetonemia.*—Diabetic coma may be recognized by the history, the sweetish odor of the breath, the glycosuria, and the subnormal temperature.

Trance.—In this condition the patient lies for several days apparently dead, the pulse and respiration being imperceptible. It is usually a manifestation of hysteria.

Somnambulism.—This is a dream-like state in which the patient performs automatically various feats, such as walking, singing, writing, etc. Mild forms, such as talking and walking in sleep, may occur in health. More marked manifestations occur in hysteria and in hypnotism.

Ecstasy.—This is a condition of apparent insensibility in which the mind is wholly absorbed with a fancy or delusion. It occurs in the hysteric. The dancing mania of the middle ages is a good illustration of it.

Catalepsy.—This is a state of motor inertia, the limbs tending to remain for long periods in any position in which they are placed. During the attacks the patient is apparently insensible to external impressions. It occurs in hysteria, hypnosis, certain psychoses (melancholia attonita), and rarely in organic brain disease.

DISTURBANCES OF THE SPECIAL SENSES.

The Eye. — *Myosis.* — Contraction of the pupil occurs in many conditions, notably in locomotor ataxia, paretic dementia, some cases of disseminated sclerosis, meningitis, cerebral tumor, old age, uremia, and opium-poisoning.

Mydriasis.—Dilatation of the pupil is also observed in many conditions, notably in atrophy of the optic nerve, paralysis of the third nerve, collapse, severe pain, epileptic seizures, hysteric attacks, belladonna-poisoning, and in some cases of locomotor ataxia and paretic dementia.

Inequality of the Pupils.—This may occur in health, in ocular defects, in organic brain disease, in paretic dementia, in locomotor ataxia, in aneurysm pressing on the cervical sympathetic, and in unilateral paralysis of the oculomotor nerve.

Argyll-Robertson Pupil.—This is one that fails to respond to light, but still accommodates for distance. It is noted especially in locomotor ataxia and paretic dementia.

Conjugate Deviation of the Eyes.—This term is applied to the forcible deflection of the eyes to one side, the visual axis still remaining parallel. It is a common symptom in gross lesions, such as hemorrhage or tumor, of the motor centers or their tracts in the brain. When the lesion is cerebral and destructive the eyes are turned away from the palsied side (toward the lesion) and when the lesion is cerebral and irritative the eyes are turned toward the convulsed side (away from the lesion). In pontine lesions the deviations are exactly reversed.

Nystagmus (*Tremor of the Eyeball*).—It may be congenital, associated with certain ocular troubles, or due to disease of basal ganglia. It is especially frequent in disseminated sclerosis and Friedreich's ataxia.

Optic Neuritis or Papillitis.—An inflammatory affection of the intra-ocular end of the optic nerve. The term "choked disk" is used to designate the condition when it is accompanied with marked swelling. Its chief causes are: Tumor of the brain, cerebral meningitis, syphilis, toxic agents (lead and alcohol), infectious fevers, anemia, and Bright's disease.

Atrophy of the Optic Nerve.—As a primary affection it is most commonly observed in locomotor ataxia and paretic dementia. Secondary atrophy results from pressure of tumors, aneurysms, etc., on the optic chiasm. Consecutive atrophy is a sequel of optic neuritis.

The Ear.—*Tinnitus Aurium* (*Noises in the Ear*).—This is observed in cerebral hyperemia and anemia, in diseases of the ear, in Ménière's disease, and after the use of certain drugs, like quinin and salicylic acid.

Hyperacusis of Hearing.—This is sometimes observed in hysteria, in facial paralysis, and in cerebral hyperemia.

Deafness generally depends upon disease of the ear itself.

PSYCHIC DISTURBANCES.

Delusion.—A delusion is a faulty belief concerning a subject capable of physical demonstration, out of which the person cannot be reasoned by adequate methods for the time being (Wood).

A *systematized delusion* is one which the patient endeavors to defend by a process of reasoning more or less logical. Systematized delusions are especially observed in monomania.

An *unsystematized delusion* is one which the patient makes no attempt to justify; he asserts his belief without reason. The majority of delusions are unsystematized, and as such are observed in most forms of insanity.

A *fixed delusion* is one which the patient retains for a considerable length of time; it is frequently systematized. Fixed delusions are observed in monomania, paretic dementia, hysteric insanity, and sometimes in melancholia.

An *expansive delusion* or a *delusion of grandeur* is one which exalts its possessor. The patient conceives that he is some noted personage, that he is worth millions of dollars, or that he is capable of performing certain marvelous feats. Expansive delusions are frequently observed in paretic dementia, mania, and hysteric insanity.

A *hypochondriacal delusion* is one which depresses its possessor. The patient believes that he has committed the unpardonable sin, that he is being persecuted, or that he is the victim of some dread disease. Hypochondriacal delusions are frequently observed in melancholia, alcoholic insanity, and in some cases of monomania and paretic dementia.

Illusion.—An illusion is a perverted perception. Thus in delirium tremens the patient may transform every piece of furniture into a demon or reptile.

Hallucination.—A hallucination is a false perception, entirely subjective, and not based upon any knowledge derived from without. An individual who hears voices and sees objects when none exist is the subject of hallucinations.

Imperative Conception.—A conception that the person knows to be false, but that, nevertheless, dominates his thoughts and often directs his actions. When he fails to recognize the falsity of his conception, it becomes a delusion.

A morbid impulse is an irresistible desire to commit an act which the patient knows to be wrong. It is usually the result of an imperative conception.

Kleptomania is a morbid desire to steal. ***Pyromania* is a** morbid desire to set fire to buildings.

Delirium.—Delirium is a mental state characterized by a rapid flight of ideas that are incoherent and often unintelligible. It may result from:

Acute Delirium (*Bell's Mania*).—A disease arising without obvious cause, and characterized by an abrupt onset, active delirium, a constant repetition of certain phrases or acts, moderate fever, often a bullous eruption, and exhaustion. It generally ends fatally in the course of a few weeks.

Mania.—In this affection the onset is not abrupt. Symptoms of impaired health and mental depression, covering a period of several weeks or months, generally precede the outbreak of the delirium.

Hysteria.—The history, age, sex, temperament, and the intermittent character of the delirium will aid in the diagnosis.

One of the Infectious Fevers.—Pneumonia and typhoid fever are especially liable to be associated with delirium. The physical signs in the former and the abdominal symptoms in the latter will usually indicate the diagnosis.

Uremia.—The urinous odor of the breath, the high arterial tension, the accentuation of the second aortic sound, and the presence of albumin and casts in the urine will suggest uremia.

Alcoholism.—The history, the appearance of the patient, the marked tremors, and frequently terrifying hallucinations will indicate alcoholism.

Inanition.—A form of delirium occasionally arises in the course of exhausting diseases. It is associated with pallor, feeble pulse, and cold extremities. It is generally of short duration, and may be recognized by the circumstances under which it develops.

DISEASES OF THE BRAIN, CORD, NERVES, AND MUSCLES.

ACUTE CEREBRAL LEPTOMENINGITIS.

Definition.—An acute inflammation of the pia mater and arachnoid.

Etiology.—(1) It may be a primary affection excited by the Diplococcus intracellularis (sporadic cerebrospinal fever) or by the pneumococcus. (2) It may be tuberculous, tubercle bacilli from a primary focus of disease elsewhere in the body reaching the meninges through the blood-vessels. (3) It may follow injury, disease of the cranial bones, or otitis media (streptococcus, staphylococcus, pneumococcus). (4) It may be a sequel of a specific fever—pneumonia, typhoid fever, diphtheria, influenza (pneumococcus, Bacillus typhosus, Bacillus diphtheriæ, Bacillus influenzæ).

Pathology.—The membranes are usually injected, cloudy, and more or less edematous. The subarachnoid space is distended with a seropurulent or purulent exudate. The substance of the brain may also be involved. The ventricles are often somewhat dilated and filled with cloudy lymph. In some the process extends over the entire and even to the spinal cord; in others it is more or less localized to the convexity or base. The tuberculous form and that following middle-ear disease are usually basilar. In the tuberculous form, which is nearly always secondary, an infiltration of yellowish, gelatinous material is found at the base, especially about the optic chiasm. Small tubercles can usually be detected along the blood-vessels in the Sylvian fissures. The amount of fluid in the lateral ventricles is often considerably increased (acute hydrocephalus).

Symptoms.—The onset may be sudden or insidious. Headache, severe and persistent, is rarely absent. Vomiting is often a prominent symptom, especially in basilar meningitis. It frequently occurs independently of the presence of food in the stomach. The temperature is moderately high (102°–104° F.) and very irregular. The pulse is generally slow (70 to 40 a minute). There are obstinate constipation and retraction of the abdomen. Irritation of the brain is soon manifested by delirium, contraction of the pupils, photophobia, intolerance to sound, general hyperesthesia, muscular twitchings, and, perhaps, convulsions.

When the exudate is sufficient in amount to exert marked pressure, paralytic phenomena develop. Palsies, gross or localized, take the place of convulsions; coma follows delirium; the pupils dilate and the eyes roll up; photophobia is replaced by blindness, and intolerance to sound by deafness. If the finger be drawn across the body, a bright-red line develops and lingers for some minutes; this is the *tâche cérébrale* of Trousseau. The pulse now becomes rapid and irregular; the respiration assumes the Cheyne-Stokes type, and the temperature falls.

When the process involves the base, retraction of the head with rigidity of the back of the neck, optic neuritis, and paralysis of the cranial nerves are prominent symptoms. The presence of pus, leukocytes, and bacteria in fluid obtained by lumbar puncture is an important sign of meningitis.

Diagnosis.—In **tuberculous meningitis** the onset is usually insidious, symptoms of ill health preceding the outbreak for days or weeks; the symptoms of a basilar involvement are marked; tuberculous lesions may be detected elsewhere in the body; tubercles are occasionally seen on the choroid, and finally tubercle bacilli may be detected in the fluid obtained by lumbar puncture.

Cerebrospinal Fever.—In this disease spinal symptoms—opisthotonos, pain in the back, and contractures—are usually marked; there may be a purpuric rash; and the fluid obtained by lumbar puncture may contain the Diplococcus intracellularis.

Typhoid Fever.—This may be recognized by the regular fever, roseolar rash, abdominal symptoms, and the Widal reaction. The severe cerebral symptoms—delirium, spasms, and retraction of the head—now and then observed in typhoid fever are usually due to cerebral congestion or to the toxemia, and only very rarely to meningitis.

Prognosis.—Very grave. Recovery never occurs in the tuberculous form, and only in very rare instances in the purulent form. The duration is from a few days to two or three weeks. When recovery does occur, blindness, paralysis, or mental impairment is apt to remain as a permanent sequel.

Treatment.—This should be conducted on the same lines as that of cerebrospinal fever. The patient should be placed in a quiet, well-ventilated room. An ice-bag should be applied to the head. In the robust, wet cups or leeches may be applied to the neck. Blisters are objectionable. The diet must be liquid. Constipation should be relieved by enemas. Restlessness, headache, and convulsions will call for bromids, chloral, phenacetin, or morphin. Koplik has reported 4 recoveries in 5 cases of meningitis due to the meningococcus, in which lumbar puncture was repeated at frequent intervals. When middle-ear disease is the exciting factor and the symptoms can be definitely localized, operation may be justifiable.

CHRONIC CEREBRAL LEPTOMENINGITIS.

Definition.—A chronic inflammation of the pia mater.

Etiology.—It may result from syphilis, alcoholism, traumatism, or sunstroke. It may be secondary to acute infectious leptomeningitis. It is an associated condition in abscess and tumors of the brain.

Symptoms.—Persistent, dull headache, mental deterioration, vertigo, muscular weakness, a low grade of optic neuritis, and occasionally nausea, vomiting, and tinnitus. Acute exacerbations are not infrequent, and are characterized by fever, severe headache, delirium, convulsions, and stupor.

Diagnosis.—**Cerebral Tumor.**—In tumor the symptoms are more severe and of a more focal character, and the optic neuritis is of a high grade.

Uremia.—This condition may be recognized by the albuminuric retinitis and the presence of albumin and casts in the urine.

Prognosis.—More or less unfavorable. A complete cure is sometimes obtained in syphilitic cases when specific treatment is instituted early.

Treatment.—In syphilitic meningitis mercury and potassium iodid should be used freely. In other instances courses of ergot and potassium bromid are occasionally useful. Applications of the thermocautery often give relief. Tonics and hypnotics are frequently indicated.

CHRONIC CEREBRAL PACHYMENINGITIS.

Definition.—Inflammation of the dura mater.

Etiology.—Inflammation of the external layer may result from injury, syphilis, or caries of the bone. Inflammation of the internal layer (hemorrhagic pachymeningitis) is most commonly met with in chronic insanities. Less frequently it follows trauma of the head or sunstroke, or occurs in chronic alcoholism, severe anemia, or chronic disease of the blood-vessels.

HEMORRHAGIC PACHYMENINGITIS.

(Hematoma of the Dura Mater.)

This condition is characterized by the formation of layers of new delicate connective tissue extraordinarily rich in thin-walled blood-vessels from which the blood is prone to escape, producing hematomata of various sizes.

Symptoms.—Often obscure. In some cases there are no manifestations during life. When the condition is marked, the following phenomena may be observed: Headache, failure of memory, impairment of intellect, stupor, contracted pupils, local convulsions, or palsies. The symptoms may alternately improve and grow worse for a long period. In grave cases, associated with extensive hemorrhagic effusion, the symptoms resemble apoplexy.

Diagnosis.—This can rarely be made with certainty.

Prognosis.—Unfavorable.

Treatment.—Grave cases should be treated as apoplexy.

CHRONIC HYDROCEPHALUS.

(Congenital Internal Hydrocephalus; Water on the Brain.)

Definition.—A condition in which there is an excessive accumulation of fluid in the ventricles of the brain.

Etiology.—The disease is either congenital or develops in the first few months of extra-uterine life. The etiology is obscure. In some cases the effusion appears to be the result of an inflammatory condition of the ventricular ependyma, while in others an occlusion of the communicating passages between the ventricles or between the ventricles and subarachnoid space seems to be the chief cause.

Pathology.—The head is large and round; the bones are thin and translucent; the sutures and fontanels are enlarged, and, if life has been prolonged, are filled with numerous Wormian bones. The convolutions of the brain are flattened and the sulci more or less obliterated. The ventricles are greatly distended with a watery fluid of low specific gravity, containing a trace of albumin. The ependyma is often thickened and roughened. Malformations are frequently observed, and probably result from the causes which induced the effusion.

Symptoms.—Sometimes the disease develops before birth, and the large head interferes with the delivery of the child. In other cases nothing peculiar is observed until the child is several months old, when the swelling of the head attracts the attention of the parents. The head assumes a globular shape; the fontanels and sutures remain open; the face becomes relatively small; the eyes protrude and are directed downward from the pressure of the fluid on the supra-orbital plates; the scalp appears thin and stretched; the superficial veins are distended; and the hair becomes scant. In some cases the head is so heavy that the thin neck can no longer support it, and it falls forward on the breast.

As a rule, the intelligence is considerably impaired, but exceptional cases are marked by precociousness. Motor phenomena are frequently present; the reflexes are exaggerated; one or more of the members may be the seat of a spastic paralysis; and convulsions develop in many cases.

The duration varies in different cases. The large majority soon die of inanition, convulsions, or some intercurrent disease to which their reduced vitality makes them an easy prey; but in a few cases life is prolonged for many years.

Diagnosis.—Hydrocephalus must not be mistaken for **rachitic enlargement of the head.** In the latter the head is square instead of globular; the intelligence is good; there are no motor phenomena; and bony enlargements are usually detected at the ends of the long bones and at the junction of the cartilages with the ribs.

Prognosis.—Unfavorable. In a few cases arrest of the disease has been spontaneous or has resulted from aspiration of the fluid.

Treatment.—The treatment is unsatisfactory. In the majority of cases, beyond dietetic and hygienic measures and the occasional use of tonics, little can be recommended. Tapping of the ventricles or of the subarachnoid space in the lumbar region (Quincke's puncture), with gradual compression of the head by means of broad strips of adhesive plaster, sometimes affords temporary relief.

PARETIC DEMENTIA.

(General Paralysis of the Insane; General Paresis; Chronic Meningo-encephalitis.)

Definition.—A chronic inflammatory affection of the cerebral cortex, characterized by a change of disposition, failure of memory, mental exaltation, delusions of grandeur, tremors, epileptiform seizures, and paralysis.

Etiology.—It occurs most frequently between the ages of thirty and fifty. It is much more common in men than in women. The exciting causes are: Prolonged mental strain, nervous shock, syphilis, alcoholism, sunstroke, and traumatism. Of these, syphilis is by far the most potent.

Pathology.—The membranes are opaque, thickened, and at places adherent to the brain substance. The cortex is more or less atrophied and increased in firmness. Microscopic examination reveals a marked increase in the neuralgia, with numerous spider cells, thickening of the vessel-walls, distention of the perivascular spaces with round cells, and degeneration of the ganglion-cells and atrophy of the nerve-fibers.

In some cases similar degenerative changes are observed in the posterior and lateral columns of the cord.

Symptoms.—The disease usually begins insidiously with a change in disposition: the industrious become slothful; the ambitious, apathetic; the chaste, dissolute; the liberal, parsimonious; the complaisant, churlish; and the truthful, false. The energy relaxes, the judgment weakens, and the memory fails. As the faculties become impaired, a peculiar egotism and a mental exaltation develop; the patient becomes boastful, loquacious, and easily provoked to furious outbreaks. The failure of memory is early noted in writing, by the use of wrong letters and the suppression of syllables. At this time motor phenomena may be observed: the tongue trembles when it is protruded; the speech is slow, hesitating, and indistinct; the pupils are often unequal; and the gait is somewhat shuffling.

The most characteristic psychic symptom of fully developed paretic dementia is the delusion of grandeur: the patient conceives that he is some distinguished personage, that he owns acres of land, or that he is the inventor of some wonderful machine. The mind is usually serene and cheerful, but periods of profound depression are not infrequent. The sensibilities are blunted, and the animal nature emphasized. The mind becomes more and more involved;

there is extreme indifference to all that transpires; the appetite is voracious, and in eating the patient bolts his food and soils his clothes. The tremor of the tongue increases, and spreads to the lips and other parts of the face; the speech is indistinct and "scanning"; the pupils fail to respond to light, but still accommodate for distance (Argyll-Robertson pupil); and the reflexes are generally increased, though they may be lost. Seizures of an epileptiform or apoplectiform character are not uncommon.

In the final stage mental power is almost obliterated; the health fails; the bladder and rectum become unretentive; the gait is more unsteady; and at last the patient is unable to leave his bed. Death usually results from exhaustion or intercurrent disease.

Diagnosis.—The insidious change in disposition, failure of memory, indistinct speech, tremors, Argyll-Robertson pupil, and delusions of grandeur are the diagnostic features.

Cerebral Syphilis.—In this disease the history, the occurrence of convulsions and of partial facial palsies, the absence of delusions of grandeur, of indistinct speech, of tremors, and the effect of treatment will usually prevent an error in diagnosis.

Prognosis.—Unfavorable. The course is not uniform: occasionally there are remissions, or lucid intervals, which last for several months or even years. The average duration is from two to three years.

Treatment.—This is chiefly hygienic and dietetic The avoidance of mental and physical excitement is imperative. When there is a suspicion of syphilis, iodids and mercurials should be given a thorough trial. As a rule, patients must be removed to asylums.

CEREBRAL PARALYSIS IN CHILDREN.

(Spastic Paralysis of Infants.)

Definition.—Hemiplegia, diplegia, or paraplegia appearing at birth or in the first few years of life, and usually associated with atrophy and sclerosis of the cerebral cortex, or porencephalus.

Pathology.—After death one of the following conditions is found: Porencephalus (cystic condition of the brain), atrophy and sclerosis of the convolutions, meningo-encephalitis, or meningeal hemorrhage.

Symptoms.—*In the hemiplegic* variety the onset is sudden, and is frequently attended with fever, convulsions, or coma. After a few hours or days these severe symptoms subside, and the child is left paralyzed on one side of the body. In rare instances the paralysis ultimately disappears and the child is restored to health, but in the large majority of cases it persists and is followed by secondary rigidity. Imbecility, epilepsy, and choreiform or athetoid movements in the affected members are very common sequelæ.

The *diplegic* or *paraplegic* form (*Little's disease*) usually dates from birth, and is characterized by rigidity and loss of power in the arms and legs, or in the legs alone. Choreiform or athetoid movements are frequently present. Children thus affected are generally idiots or imbeciles. Meningeal hemorrhage, induced by tedious labor or the use of the forceps, appears to be chiefly responsible for this variety.

Treatment.—During the convulsive stage an ice-bag should be applied to the head, and chloral or bromid administered by the mouth or rectum. The paralysis resists treatment; but subsequent rigidity may be lessened by massage and passive movements, and the deformity by mechanical appliances.[1]

CEREBRAL HYPEREMIA.

(Congestion of the Brain.)

Etiology.—*Acute congestion* results from exposure to the sun; from the use of certain drugs, like alcohol and nitroglycerin; from excessive brain-work; or from some reflex disturbance, as gastric irritation.

Chronic congestion results from some local obstruction to the return of blood from the brain, as by a tumor in the neck; from obstruction to the general circulation, as in chronic heart and lung disease; from the suppression of some habitual discharge, as the menstrual flow at the menopause; or from some general cause, such as prolonged anxiety, overwork, excesses, irregular living, etc.

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[1] The foregoing description is based upon Osler's elaborate monograph.

Symptoms.—*Acute Form.*—The chief symptoms are: Intense headache; vertigo; intolerance to light and sound; restlessness; tinnitus aurium; and sleeplessness, or sleep disturbed by horrible dreams.

Chronic Form.—This is characterized by vertigo; dull headache; failure of memory; irritability; inability to concentrate the thoughts; and disturbed sleep. The symptoms grow worse when the recumbent posture is assumed. Ophthalmoscopic examination reveals retinal hyperemia. In marked cases there may be exacerbations closely resembling apoplexy, in which there is unconsciousness, followed by temporary paresis.

Prognosis.—The prognosis depends on the cause; when this can be removed, the prognosis is favorable.

Treatment.—*Acute Congestion.*—The patient should be placed in a darkened, well-ventilated room. The head and shoulders should be slightly elevated. An ice-bag should be applied to the head. In some cases leeches or wet-cups may be applied to the neck. Sedatives like bromid of potassium and aconite sometimes do much good. Hot foot-baths are often serviceable. If there is constipation, it should be relieved by a brisk saline purge.

In *chronic cases* the cause should be ascertained, and, if possible, removed. The habits of the patient must be regulated. The diet must be light and nutritious. Constipation must be relieved by diet or by the occasional use of a saline laxative. Sedatives like bromid of potassium and aconite are useful. In the apoplectiform attacks venesection is indicated.

CEREBRAL ANEMIA.

Etiology.—General cerebral anemia as a *chronic affection* may result from cardiac disease, especially aortic stenosis. It may be associated with general anemia. It may be due to atheromatous obstruction of the arteries.

Overwork, prolonged emotional excitement, irregular living, and excesses are also said to predispose.

As an *acute condition* it exists in syncope and shock; after hemorrhage; after the sudden withdrawal of fluid from the abdominal cavity; and after ligation of the carotid artery.

Symptoms.—*Acute Form.*—Pallor of the face, vertigo, confusion of ideas, ringing in the ears, dimness of vision, dilatation of the pupil, nausea, and a tendency to yawn. In extreme anemia there may be convulsions and coma.

The *chronic form* is characterized by vertigo, headache, disturbed sleep, intolerance to light and sound, irritability of temper, failure of memory, inability to concentrate the attention on one subject, a tendency to syncope, and extreme lassitude. The symptoms improve when the patient lies down.

Diagnosis.—Cerebral anemia closely simulates *cerebral congestion*, but in the latter there is no tendency to syncope; the symptoms grow worse when the patient lies down; the pupils are contracted instead of dilated, and the urine is apt to be decreased.

Prognosis.—The prognosis depends on the cause; when this can be removed, the prognosis is favorable.

Treatment.—In acute cases diffusible stimulants like nitroglycerin, ammonia, and alcohol are indicated. In chronic cases the cause should be ascertained, and, if possible, removed. When it is due to general anemia, iron, arsenic, and quinin are useful remedies. When dependent on valvular disease, rest and the use of digitalis, strophanthus, or strychnin are the remedial measures.

CEREBRAL HEMORRHAGE.

(Cerebral Apoplexy.)

Etiology.—The affection is most commonly met with in persons over forty, in whom the blood-vessels are atheromatous, but it may occur in childhood or infancy. All causes that lead to degeneration of the arteries, such as rheumatism, gout, syphilis, alcoholism, and Bright's disease, predispose to it. Sufferers from chronic Bright's disease are very liable to die of apoplexy on account of the association of cardiac hypertrophy with arterial degeneration. Heredity predisposes, inasmuch as members of certain families are particularly prone to sclerosis of the vessels. The attack may be precipitated by mental or physical excitement, alcoholic excess, or some reflex disturbance, as gastric irritation.

In children it may be excited by a paroxysm of whooping-cough or by a convulsion. Occasionally, it occurs after diphtheria or scarlet fever, the toxins of these diseases producing degenerative changes in the arterial walls.

Pathology.—In children the hemorrhage is most commonly cortical; in adults it is usually within the brain-mass. The blood-vessels are generally atheromatous, and are sometimes the seat of miliary aneurysms. The hemorrhage varies greatly in quantity: sometimes it is small—merely a capillary oozing; at other times it may flood an entire hemisphere. Its most common seat is the internal capsule—the motor highway between the optic thalamus and the corpus striatum. In recent hemorrhages the clot is dark and soft, and the surrounding tissue stained and more or less lacerated. If the hemorrhage has not been very copious, the clot loses its color, shrinks, and is finally absorbed, and the damaged cerebral fibers are replaced by proliferated connective tissue, which contracts and forms a scar more or less pigmented with hematoidin. In other cases, instead of a scar, a cyst is formed that incloses a clear, straw-colored fluid.

Large effusions are followed by secondary changes, which are systemic and extend in the direction in which the affected nerve-fibers transmit impulses; that is, toward the periphery, if the fibers are motor, and toward the nerve-centers if they are sensory. After an extensive lesion of the internal capsule secondary degeneration of the motor tracts soon begins and may be traced downward into the spinal cord.

Symptoms.—*Prodromal symptoms* indicating cerebral congestion frequently precede the attack; these are headache, vertigo, disturbed sleep, tinnitus aurium, and, perhaps, a sense of numbness or weakness on the side that is to be affected. Persistent vomiting sometimes precedes the hemorrhage.

The Attack.—In many cases the patient falls suddenly unconscious without previous warning. The face is flushed; the eyes are injected; the lips are blue; the breathing is stertorous; the pulse is full and slow; the temperature is at first subnormal from shock, but later it is elevated from irritation; and the urine and feces may be passed involuntarily. Convulsive seizures are not infrequent; they result from irritation transmitted to the undamaged motor regions. Even while the patient is comatose, the paralysis may be detected. The head and eyes may be strongly rotated toward the side of the hemorrhage (conjugate deviation); one cheek often flaps more than the other; the pupils may be unequal; any movements that the patient may make are restricted to the sound side; when the affected arm is raised and let fall, it drops lifeless; and occasionally the temperature is higher in the axilla of the paralyzed side. In grave cases the patient does not awake from the coma; the pulse grows feeble; the respirations assume the Cheyne-Stokes type; the reflexes are abolished; mucus collects in the throat and produces a rattling sound; the temperature rises to 103° to 104°; and death results after the lapse of a few hours or one or two days.

In certain cases the paralysis rapidly sets in, but unconsciousness develops gradually and does not become complete for twenty-four hours (ingravescent apoplexy). In other cases of cerebral hemorrhage loss of consciousness is very transient or wholly wanting.

Subsequent Symptoms.—When the attack does not prove fatal, consciousness is usually restored in from twelve to forty-eight hours, and if the hemorrhage is in its usual location, there remains a hemiplegia on the opposite side. The muscles of the upper part of the face and thorax, however, usually escape, because they are accustomed to act in unison with their fellow on the opposite side, and such muscles appear to be innervated from both sides of the brain. When the tongue is protruded, it deviates toward the paralyzed side. The deep reflexes are exaggerated on the affected side and tickling the sole of the foot causes extension of the great toe (Babinski's sign). Aphasia is common with right hemiplegia. There is no tendency to rapid wasting of the affected muscles. Sensation is unimpaired unless the posterior limb of the internal capsule is also involved, when there is hemianesthesia with the hemiplegia. The gait is peculiar; in walking the patient supports the paralyzed arm and swings the leg forward by a rotary movement imparted to it by the trunk. When the clot has been small, the paralysis may completely disappear. More frequently, recovery is only partial. The power of the facial muscles is generally restored entirely, and the leg usually improves more than the arm.

In unfavorable cases the muscles become rigid from a degenerative process traveling down the direct and crossed pyramidal tracts of the spinal cord; this condition is indicative of permanent disability.

Mental symptoms, especially impairment of memory and loss of emotional control, frequently follow the attack.

Diagnosis.—The coma of apoplexy must be distinguished from **uremia, opium-poisoning, alcoholism,** and **sunstroke.** The age of the patient; the condition of the arteries; the evidence of paralysis; the difference of temperature in the two axillæ; and the absence of other cause will usually prevent an error in diagnosis.

Embolism.—This usually occurs in earlier life; it is commonly associated with valvular disease; premonitory symptoms are rarely present; the pulse is more often weak than strong; disturbances of temperature and breathing are less marked.

Thrombosis.—This also produces hemiplegia; but its development is usually gradual; unconsciousness is often absent, and temperature and breathing are not much disturbed.

Hemiplegia from Other Causes.—*Tumors and abscess in the brain* may produce hemiplegia, but the latter develops gradually and is usually associated with other cerebral phenomena, such as persistent headache, vertigo, ocular palsies, choked disk, etc.

Hysteric Hemiplegia.—In hysteria the face escapes; there is frequently anesthesia on the affected side; the gait is peculiar, in that the patient pushes the paralyzed limb instead of swinging it. These features, together with the age, temperament, sex, and mode of onset, will usually suggest the true cause.

Prognosis.—Always doubtful. Persistent and complete unconsciousness, high temperature, loss of reflexes, and embarrassed respiration are unfavorable phenomena. When the attack does not prove fatal, there is always danger of recurrence, since the etiologic conditions still remain.

Treatment.—*Prophylaxis.*—Patients predisposed to apoplexy should lead a quiet life, free from mental and physical excitement. The diet should be nutritious, but easily digestible. Constipation should be relieved by the occasional use of a saline laxative. To secure a free return of the blood from the brain the clothes at the neck should be loose.

The Attack.—The head and shoulders should be slightly elevated, and an ice-bag applied to the head. Croton oil (1 to 3 drops) in a little glycerin or olive oil may be placed on the back of the tongue to secure prompt catharsis. If the pulse is strong, venesection is indicated and should be continued until the pulse softens. Bleeding cannot undo the damage already done, but by relieving cerebral congestion, it may serve to arrest bleeding that is still in progress or to prevent an early recurrence. On the other hand, when the face is pale and the pulse feeble, stimulants, like ammonia, ether, and camphor, should be given very cautiously. When collections of mucus interfere with breathing, the patient should be gently turned on his side and the mucus removed

To prevent the formation of bed-sores the position should be frequently changed and the parts subjected to pressure thoroughly cleansed.

Subsequent Treatment.—Even in the mildest cases the patient should not be allowed to leave his bed for two or three weeks, and during this time the diet should be light and unstimulating. After the acute symptoms have entirely disappeared, which will rarely be earlier than ten days or two weeks after the attack, massage should be systematically practised. It aids in the restoration of power and in the prevention of contractures. After the lapse of three or four weeks triweekly applications of the faradic current may be of service. Strychnin is often given at this time, but it probably exerts no other influence than that of a general tonic. In some cases warm saline baths (90°–95° F.) combined with passive movements prove useful adjuvants.

OBSTRUCTION OF THE CEREBRAL ARTERIES.

(Embolism; Thrombosis.)

Etiology.—*Cerebral emboli* may be derived from the valves of the heart in endocarditis; from an atheromatous plate in the aorta; or from thrombus in the heart or in the sac of an aneurysm. Obstruction from embolism may occur at any age, but it is far more commonly observed in young adults than at the extremes of life.

Cerebral thrombi are usually caused by atheroma or syphilitic endarteritis. They are usually observed in advanced years, but those dependent on syphilis frequently occur in early adult or middle life.

Pathology.—*Emboli* are most frequently found in a branch of the left middle cerebral artery. When the artery obstructed is a large one, infarction of the brain with softening ensues. If the area affected is small, absorption of the dead tissue usually follows, with the formation of a cicatrix. Infective emboli give rise to abscesses.

Thrombi are usually found in the basilar, middle cerebral, or vertebral arteries, and produce the same lesions as emboli.

Symptoms.—*An embolus* lodging in the middle cerebral artery usually causes abrupt hemiplegia and frequently aphasia. There may be no prodromes, and consciousness may be preserved during the seizure.

When the basilar artery is obstructed, there may be extensive paralysis on both sides of the body, and later symptoms of bulbar disease—namely, paralysis of the lips, pharynx, and esophagus, disturbance of the heart, and Cheyne-Stokes breathing.

In *thrombosis* the symptoms are similar to embolism, but they develop more slowly, and are frequently preceded by prodromes indicating disturbed cerebral circulation, such as headache, vertigo, disturbed sleep, failure of memory, numbness and tingling in the limbs to be affected. There is a marked tendency to recurrence of attacks.

Subsequent Symptoms.—In both embolism and thrombosis, if the artery obstructed has been large, the paralysis is likely to persist and to be followed by symptoms of cerebral softening—namely, failure of memory, vertigo, headache, disturbed sleep, irritability, and finally dementia.

Diagnosis.—The differential diagnosis between cerebral embolism or thrombosis and **hemorrhage** has already been considered (see p. 393).

Prognosis.—The prognosis is always grave; unless the symptoms are slight, complete recovery is rare.

Treatment.—After obstruction from embolism the patient should be kept at absolute rest for a week or two, and subsequently the paralysis treated as after apoplexy. In thrombosis treatment is rarely of avail; in syphilitic cases, however, active antiluetic treatment should be instituted.

MORBID GROWTHS IN THE BRAIN.

(Tumors of the Brain.)

Etiology.—The etiology of brain tumors is obscure. Males are more frequently affected than females. No age is exempt, but the majority of brain tumors occur between the ages of thirty and fifty. Occasionally the history of some remote injury is obtainable. Heredity predisposes to the extent that it favors the development of tubercle and cancer.

Varieties.—Tubercle, gumma, glioma, aneurysm, cysts, sarcoma, and carcinoma are the most common varieties. Less frequently fibroma, psammoma, and lipoma are observed.

Pathology.—*Tuberculous tumors* or *tyromata* vary in size from that of a pea to that of an egg; they may be single or multiple, and are usually observed in the young.

Gumma.—This appears as a round, yellow, caseous mass, and is nearly always on the surface of the brain, into which it grows from the overlying membranes. It is usually met with between the ages of thirty and forty.

Glioma.—This tumor is found almost exclusively in the brain. It arises from the neuroglia, and may be soft, like brain-substance, or firm, like fibrous tissue. It is chiefly met with in the young.

Aneurysm.—Encephalic aneurysm may be single or multiple. Miliary aneurysms of small vessels frequently excite apoplexy. The most common seats of large aneurysms are the middle cerebral, basilar, and internal carotid arteries.

Cysts.—These are usually congenital (porencephalus) or result from hemorrhage, but sometimes they result from the Tænia echinococcus (hydatid cyst) or Tænia solium (Cysticercus cellulosæ).

Sarcoma.—This is usually a circumscribed tumor, and commonly grows from the membranes. It is generally primary.

Carcinoma.—This is nearly always secondary and multiple.

Symptoms. — *General Symptoms.* — (1) Headache is rarely absent; it is sometimes localized and associated with tenderness on pressure. (2) Vomiting is a common symptom, especially in tumors of the base of the brain; it is often unassociated with nausea, and does not relieve the attending headache. (3) Optic neuritis or optic atrophy is present in about 80 per cent. of the cases. (4) Vertigo is often marked, especially in tumors of the basal ganglia and cerebellum. (5) Convulsions, local (Jacksonian epilepsy) or general, occur in about 50 per cent. of all cases. (6) Psychic phenomena—failure of memory, depression of spirits, irritability of temper, and emotional states—are not infrequently present. Insomnia, changes in the rate and rhythm of the pulse, polyuria, and glycosuria are occasional symptoms.

Focal Symptoms.—These depend entirely upon the location of the tumor. The following are the chief localizing symptoms:

Prefrontal Region.—Mental torpor, irritability, and drowsiness deepening into stupor frequently appear. Motor agraphia and aphasia may result from compression of the second and third frontal convolutions.

Motor Region (the Ascending Frontal Convolution).—When the tumor irritates the centers, local convulsions develop; when it exerts enough pressure to destroy function, paralysis results.

Posterior Portion of the Third Frontal Convolution (Left Side).—Motor or ataxic aphasia is a characteristic symptom.

Temporal Lobe, First and Second Convolutions (Left Side).—Tumors in this region cause word-deafness.

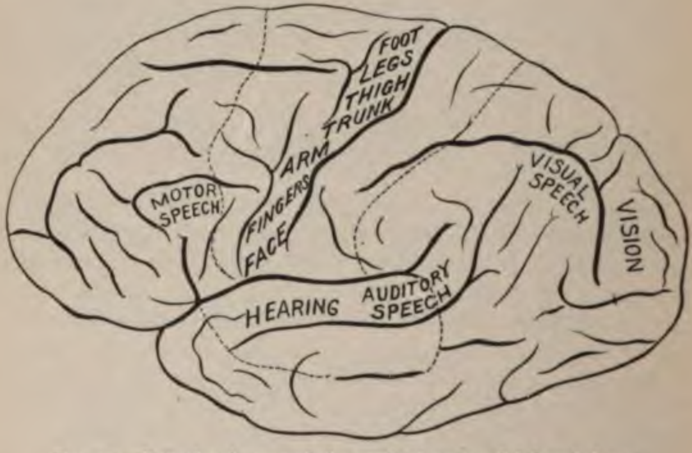

FIG. 18.—Functional areas of the cerebral cortex, left hemisphere.

Angular and Supramarginal Gyri (Left Side).—Word-blindness and apraxia usually develop.

Parietal Lobe.—Tumors in this region frequently occasion disturbances of cutaneous and muscular sensibility, especially a loss of power to recognize the shape of objects by touch (astereognosis).

Occipital Lobe.—Hemianopsia is common, and there may be psychic blindness if the growth is on the left side. Word-blindness may also result from pressure on the angular gyrus.

Internal Capsule.—Lesions of the middle third cause hemiplegia on the opposite side; of the posterior third, hemianesthesia of the opposite side.

Corpus Striatum.—Large lesions produce hemiplegia from pressure on the internal capsule.

Optic Thalamus.—Large lesions may produce hemianesthesia from pressure upon the posterior limb of the internal capsule and sometimes hemianopsia.

Corpora Quadrigemina.—There may be incoördination, oculomotor palsies, nystagmus, and loss of the pupil-reflex.

Crus Cerebri.—Tumors in this locality cause paralysis of the third nerve on the side of the lesion and hemiplegia on the other side.

Pons.—Pontile growths may occasion hemiplegia and hemianesthesia on the side opposite to the lesion, and paralysis of the cranial nerves, especially of the facial, on the side of the lesion.

Corpus Callosum.—Tumors in this region may cause mental symptoms,—stupor, irritability, hallucinations, attacks of excitement,—followed by motor paralysis.

Cerebellum (Middle Lobe).—The characteristic symptoms are headache, staggering gait, vertigo, and vomiting. Paralysis may result from pressure on the pyramidal tracts.

Diagnosis.—*The Character of the Growth.*—This cannot always be determined. The early age, the rapid progress, and the family history may suggest *tubercle*. The early age, slow progress, and mild pressure-symptoms may suggest *glioma*. The history, age, and concomitant symptoms will indicate *syphilis*. The presence of a primary growth will lead to the diagnosis of *cancer*. The presence of a thrill, bruit, and marked tinnitus and the absence of optic neuritis would suggest aneurysm.

Abscess.—Cerebral tumor must be distinguished from abscess. The latter usually results from traumatism or is secondary to a focus of suppuration in some other part of the body; its progress is usually more rapid; optic neuritis is less common; and there is often febrile disturbance, with leukocytosis.

Chronic Meningitis.—In this affection the symptoms indicate a diffuse lesion; disturbances of temper, memory, and sleep are more marked; optic neuritis is not frequent.

Prognosis.—Always grave. When the tumor is not gummatous and is not suitable for operative interference, the prognosis is absolutely unfavorable. The duration is from a few months to several years.

Treatment.—As there is always a possibility that the tumor is syphilitic, mercury and iodids should be tried in every case the nature of which is in the least uncertain. Operative interference must be considered when the tumor is localized and situated in the cerebral cortex or cerebellum. Less than ten per cent. of brain tumors are open to operation. Growths in the motor region offer the best chances of success.

In inoperable nonsyphilitic growths treatment is palliative. Cold applications to the head, bromids, antipyrin, and morphin are required to relieve pain.

ABSCESS OF THE BRAIN.

(Suppurative Encephalitis.)

Etiology.—(1) It may be traumatic. (2) It may be secondary to suppurative inflammation of adjacent parts, as caries of the temporal bone following otitis media. (3) It may be secondary to some distant focus of suppuration, as in pulmonary abscess, hepatic abscess, ulcerative endocarditis. (4) It may follow one of the infectious fevers.

Pathology.—The abscess varies in size from a pea to one large enough to fill an entire hemisphere. The surrounding tissues are hyperemic, edematous, and more or less infiltrated. In the acute form the abscess is diffuse, but in long-standing cases the pus is encapsulated by a thick fibrous sac. The temporosphenoid lobe and the cerebellum are the most frequent seats. Abscesses secondary to distant foci of suppuration are commonly multiple.

Symptoms.—Abscesses following injury frequently run an acute course, and are characterized by high fever, rigors, headache, delirium, convulsions, vomiting, and coma.

In chronic cases the *general symptoms* are headache, tenderness of the head to percussion, irritability, mental impairment, vertigo, vomiting, stupor, pallor, and loss of flesh and strength. The temperature is variable; it may be elevated but not rarely it is normal or subnormal. The *focal phenomena* vary with the location of the abscess. Involvement of the motor area may be attended with convulsions or paralysis in one limb; of the temporosphenoid lobe, with deafness and perhaps aphasia; of the occipital lobe, with hemianopia; of the cerebellum, with persistent vomiting and loss of coordination.

Prognosis.—Grave. When the focal symptoms indicate involvement of an accessible region like the motor area, temporosphenoid lobe, or cerebellum, operative interference affords considerable hope of success.

Treatment.—When the abscess is located in one of the regions specified, the skull should be trephined and the pus evacuated. In other cases the application of wet cups to the neck, of ice-bags to the head, and the internal use of opium, bromid of potassium, or of chloral may temporarily relieve the distress.

APHASIA.

Definition.—A failure of word-memory; an inability to utter words, to write them, or to comprehend them.

Varieties.—Motor and sensory.

Motor Aphasia.—*Ataxic Aphasia.*—Impairment or loss of articulate speech from an inability to recall the efforts needed to pronounce words rather than from muscular paralysis. It is the most common form of aphasia. The lesion is in the posterior part of the third left frontal convolution (Broca's region).

Motor Agraphia.—In this condition the patient, though able to read, is unable to write voluntarily or from dictation, or to copy. The lesion is in the posterior part of the midfrontal convolution (?).

Paraphasia.—Misplacement of words and syllables. The patient talks jargon, though he understands what he hears and sees and can articulate clearly. The lesion is in one of the association tracts between the speech centers.

Sensory Aphasia.—*Word-blindness* (*Alexia*).—Inability to recognize written or printed words. The lesion is situated in the angular and supramarginal convolutions of the left side. The patient being unable to call to mind the appearance of words, may also lose the power to write spontaneously (sensory agraphia). Word-blindness is usually associated with hemianopsia, as a lesion of the angular gyrus is very apt to involve the underlying visual tracts.

Word-deafness.—Inability to recognize spoken words. The lesion is in the middle of the first and second temporal convolutions of the left hemisphere. Though the patient may be able to read, he is often unable to speak correctly, since he cannot recall the sound of the words.

Apraxia.—This is a loss not only of word memories but of all memories having to do with sight, hearing, and touch. The patient is unable to recognize objects when he sees, hears, or touches them. When the loss of concepts is complete there is also the inability to employ objects in the proper way. Apraxia is usually associated with aphasia. The following are the chief varieties:

Psychic Blindness.—This is a condition in which objects are seen, but not recognized. It results from a lesion of the occipital lobe and is usually associated with hemianopsia.

Psychic Deafness.—This is a condition in which not only words, but all sounds, though heard, awaken no intelligent conception. The bark of a dog, for instance, calls to mind no visual image of the animal. Psychic deafness, like word deafness, results from a lesion of the left temporal convolutions.

Astereognosis.—A loss of the power of recognizing objects by touch. It is a symptom of disease in the middle portion of the posterior central convolution and adjacent part of the inferior parietal lobule.

Pathology.—The lesions that produce aphasia are manifold; the most important are: Tumor, gumma, abscess, depressed fracture, embolism, thrombus, or softening in the localities that correspond to the various forms of aphasia. In right-handed subjects the lesion is on the left side of the brain; in the left-handed it may, however, be on the right side. Aphasia is not always due to organic disease; it may be noted in congestion of the brain, in sudden fright, in the convalescence of fevers, in migraine, after epileptic seizures, and in hysteria.

Diagnosis.—Aphasia must be distinguished from **aphonia.** The latter condition is an inability to utter sounds, a power not lost in aphasia; moreover, aphonia is generally dependent upon some abnormality of the larynx or of the nerves leading thereto.

Prognosis.—This depends entirely on the cause. After apoplexy the prognosis should be guarded. In cerebral softening it is absolutely unfavorable. When aphasia develops in the young, the outlook is much more hopeful.

Treatment.—The causal condition will require attention. The patient may be instructed to speak and to interpret after the manner employed in teaching the young.

SPINAL LEPTOMENINGITIS.

(**Spinal Meningitis.**)

Definition.—An inflammation of the spinal pia mater.

Etiology.—Acute spinal leptomeningitis usually occurs as a part of cerebrospinal meningitis. As a primary disease, without involvement of the cranial meninges, it is rare. It occasionally follows one of the infectious fevers, traumatism, or exposure. In some instances it is tuberculous.

Pathology.—*Acute Form.*—The membranes are opaque, thickened, congested, and adherent. The fluid in the arachnoid space is increased. In very acute cases there is more or less purulent infiltration. The periphery of the cord is always involved.

Chronic Form.—The membranes are very thick and fused into one homogeneous fibrous mass.

Symptoms.—*Acute Form.*—The disease may begin with a chill, followed by a moderate fever. There is intense pain in the back, radiating along the course of the nerves. The back is tender. The spinal muscles are rigid and contracted—sometimes so much so as to induce opisthotonos. The reflexes are increased. When the exudate is sufficient to make considerable pressure on the cord, paralytic phenomena develop, such as slight anesthesia and paresis of the limbs.

There are no cerebral symptoms unless the meninges of the brain are involved.

Diagnosis.—**Myelitis.**—In this affection the pain is less severe; there is less tendency to spasm; paralysis and anesthesia are more marked; the bladder and rectum are early involved; and the formation of bed-sores is common.

Tetanus.—The presence of a wound; the absence of fever; the early involvement of the jaw; and the absence of marked tenderness in the back will suggest tetanus.

Prognosis.—Extremely grave. Recovery sometimes follows, but rarely without partial paralysis.

Chronic Leptomeningitis.—This is characterized by pain in the back; stiffness of the muscles; hyperesthesia and paresthesia of the limbs, but rarely anesthesia; some loss of power; and exaggerated reflexes.

Treatment.—This is the same as that of cerebrospinal meningitis.

CHRONIC SPINAL PACHYMENINGITIS.

(Cervical Hypertrophic Pachymeningitis; Internal Pachymeningitis.)

Definition.—A chronic inflammatory affection of the dura mater, characterized by severe pains in the head, shoulders, arms, and loins, followed by paresis, wasting, and anesthesia.

Etiology.—Prolonged exposure to cold, spinal concussion, alcoholism, and syphilis are predisposing factors. It may be secondary to inflammation of neighboring structures, such as the vertebræ in Pott's disease.

Pathology.—The membranes are thickened, opaque, and adherent; the vessels are dilated; and the spinal fluid is increased. In advanced cases the membranes are glued together and form a thick, homogeneous, fibrous mass. The cervical region is most commonly affected. The inflammation may extend to the cord and peripheral nerves.

Symptoms.—These include sharp pains radiating into the head, shoulders, arms, and loins, and paresthesia, followed by loss of power, anesthesia, wasting, and rigidity, particularly in the upper extremities. When the lower part of the cord is involved, the same phenomena are observed in the legs, and the knee-jerk is increased. The duration of the disease is several years.

Diagnosis.—**Chronic Poliomyelitis.**—The absence of pain and of anesthesia will separate poliomyelitis from pachymeningitis.

Multiple Neuritis.—In this affection the pain is less marked in the back and more marked in the extremities, and the nerve-trunks are tender on pressure.

Syringomyelia.—In this affection there is much less pain and tactile sensation is preserved.

Prognosis.—This depends on the extent and cause. When the involvement is slight or is due to syphilis, the prognosis should be guardedly favorable.

Treatment.—Counterirritation should be made along the cord by frequent blisters or the actual cautery. Iodid of potassium may be administered for its absorbent effect, and in syphilitic cases it should be given freely in conjunction with some mercurial.

ACUTE MYELITIS.

Definition.—An acute inflammation of the substance of the cord, characterized by marked disturbances of motion, sensation, and nutrition.

Varieties.—When only a limited vertical area of the spinal cord is involved, the condition is termed *transverse myelitis.* When a large vertical section is affected, the disease is termed *diffuse myelitis.*

Etiology.—Traumatism, exposure, or overexertion may induce it. It may be a sequel of syphilis or of an acute infectious disease, such as smallpox, typhoid fever, pneumonia, dysentery, or gonorrhea. It is sometimes secondary to caries of the spine or tumors of the cord.

Pathology.—The membranes are usually injected and opaque. The substance of the cord is red and soft, and the line of demarcation between the gray and white matter is indistinct. In very acute cases the substance of the cord may flow out as a reddish, creamy fluid when the membranes are cut. Occasionally there are conspicuous hemorrhagic effusions (hematomyelitis).

Microscopically, the bloodvessels are found to be greatly dilated and surrounded by leukocytes; the myelin sheaths swollen and fatty; the axis-cylinders swollen, granular, and perhaps disintegrated; and the nerve-cells necrotic, vacuolated, and in places without processes. In the severe cases in which the cord is reduced to a puriform mass examination reveals only fat droplets, pigment particles, granular cells, leukocytes, and detritus.

Symptoms.—**Acute Transverse Myelitis.**—The onset is frequently marked by pains in the back, a girdle sensation, and numbness in the limbs. Paralysis and anesthesia of the parts below the lesion quickly develop. The control of the bladder and rectum is lost. Bed-sores are apt to form over the sacrum and heels. Of the paralyzed muscles only those supplied by nerves arising from the diseased segment undergo atrophy and yield reactions of degeneration. The knee-jerks are usually increased and the legs stiff and rigid. There may be a loss of tendon reflexes with flaccidity of the muscles, however, if the cord is entirely divided (Bastian) or if the lesion invades the lumbar segments. There is often a zone of hyperesthesia just above the level of the anesthesia. Fever may or may not be present.

The outlook is always serious. More or less disability usually remains after the subsidence of the inflammation. Complete recovery may occur, however, in mild cases. Not rarely death ensues from cystitis and pyelonephritis, bed-sores and sepsis, or pneumonia.

Acute Central Myelitis.—This resembles the former, but the trophic disturbances are much more marked and the duration is shorter. The disease is characterized by moderate fever and its associated phenomena, pain in the back, complete loss of power and of sensation, loss of reflexes, incontinence of urine and feces, rapid wasting of the muscles, and the early development of bed-sores. The disease frequently proves fatal in from one to two weeks.

Diagnosis.—**Acute Poliomyelitis.**—In this disease the bladder and rectum are not involved and there are no sensory disturbances.

Landry's Disease or Acute Ascending Paralysis.—In this affection trophic disturbances are absent; the bladder and rectum are not involved; and the loss of sensation is slight.

Multiple Neuritis.—The "girdle pain" is absent; the sphincters are not affected; bed-sores are rare; and pain is more marked in the extremities than in the back.

Meningitis.—The girdle pain is absent; the sphincters are not affected; the irritative phenomena are more marked than the paralytic.

Hemorrhage into the Cord.—The paralysis develops abruptly.

Prognosis.—Always extremely grave. Acute central myelitis is invariably fatal. In other cases recovery attended with partial paralysis occasionally follows.

Treatment.—If possible, the patient should be placed on a water-bed or air-bed. Counterirritation should be avoided, on account of the danger of bed-sores. Cold, however, in the form of Chapman's ice-bags, may be applied to the spine. Daily warm baths (90° F.) lasting about ten minutes are useful.

Every precaution must be taken against the development of bed-sores. Frequent change of the patient's position, absolute cleanliness of the parts subjected to pressure, and bathing with alcohol and water will do much toward obviating this complication. Retention of urine must be met by systematic catheterization under the most strict antiseptic precautions. When there is constant incontinence, a carefully adjusted urinal should be employed.

Any tendency to cystitis will call for daily irrigation of the bladder with a solution of boric acid or other mild antiseptic solution. If recovery with partial paralysis result, massage and electricity may aid in bringing back some of the lost power.

CHRONIC MYELITIS.

Etiology.—Traumatism, exposure, alcoholism, and syphilis are predisposing factors. It is sometimes induced by the extension of inflammation from adjacent structures—meninges and vertebræ. It is occasionally a sequel of acute myelitis.

Pathology.—The membranes are opaque and adherent. The whole cord has a grayish color; it is firmer than normal and somewhat contracted.

Microscopic examination reveals destruction of nerve elements and their replacement by an overgrowth of neurogliar tissue.

Symptoms.—The disease begins gradually with numbness, tingling, or burning in the lower extremities, followed by a loss of power and sensation. The reflexes are generally exaggerated. The sphincters soon become involved. The muscles do not waste until the disease is far advanced. As in other organic affections of the cord, there is often a sense of constriction, or "girdle pain," at the level of the disease. The disease progresses very slowly, the duration being from six months to ten years.

Diagnosis.—The diagnosis rests on the gradual development of symptoms indicating a general involvement of the cord.

Treatment.—Prolonged rest is desirable. Daily warm baths are grateful. Counterirritation in the form of light touches of the actual cautery is useful in relieving pain, but it does not seem to exert any direct influence on the progress of the disease. When there is reason to suspect the existence of syphilis, mercury and the iodids should be given a thorough trial. Tonics are often indicated. Massage, passive movements, and electricity are useful in maintaining the nutrition of the affected muscles. Early measures should be taken to prevent the formation of bed-sores and the development of cystitis.

ACUTE ANTERIOR POLIOMYELITIS.

(Infantile Paralysis; Atrophic Spinal Paralysis.)

Definition.—An acute disease, occurring almost exclusively in young children, characterized anatomically by a destruction of the ganglion-cells in the anterior gray horns of the cord, and manifested clinically by abrupt paralysis and rapid wasting of certain muscles.

Etiology.—The greatest number of cases occur within the first three years, and the disease is far more common in summer than in winter. The sudden onset, the absence of any known exciting cause, and the fact that it has occurred endemically suggest an infectious origin.

Pathology.—Microscopic examination of the cord in early cases reveals great distention of the vessels, cellular infiltration of the perivascular spaces, and degeneration and disintegration of the ganglion cells. These lesions are probably due to occlusion of certain branches of the anterior spinal artery by thrombi of infective origin.

In old cases the lesions found consist of a marked atrophy of the anterior horn, an absence of ganglion cells, and increase of the neuroglia.

The motor nerve-fibers corresponding to the diseased segment and the muscle-fibers innervated by these nerves also show degenerative changes.

Symptoms.—Generally the onset is abrupt; often the child is put to bed in apparent health and in the morning is found paralyzed in one or more limbs. In some cases febrile symptoms precede the attack, and more rarely the disease is ushered in with a chill, a convulsion, or delirium.

The paralysis at first may be quite extensive, but more commonly it confines itself to certain groups of muscles in the upper or lower extremities. The latter are especially prone to suffer; the affected muscles are relaxed, and the surface is cold and often cyanosed. The paralysis is peculiar in its irregular distribution and in its tendency to improve spontaneously up to a certain limit. There are no sensory disturbances, no involvement of the bladder and rectum, and no tendency to bed-sores. The muscles that are permanently affected rapidly waste and ultimately yield the reactions of degeneration. Permanent deformity often ensues from the ratardation of growth in the paralyzed limb and the occurrence of contractures in the unaffected muscles.

Diagnosis.—The abrupt onset will distinguish it from both the **muscular dystrophies** and **chronic poliomyelitis.** The absence of sensory disturbances, bed-sores, and paralysis of the bladder and rectum will distinguish it from **myelitis.** The presence of cerebral symptoms and of exaggerated reflexes, and the absence of reactions of degeneration and of early wasting will distinguish **cerebral paralysis of childhood** from acute poliomyelitis.

Prognosis.—Unless the initial symptoms are very severe, the prognosis as regards life is good. In all cases some of the paralysis disappears. Occasionally the improvement is so great that the usefulness of the member is not impaired; but far more frequently the residual paralysis is sufficient to cause considerable deformity and disability.

Treatment.—During the acute stage the child should be confined to bed. Mild laxatives and febrifuges may be used with some advantage. Ergot is often given with the view of lessening congestion, but it is of doubtful utility. The affected limbs should be wrapped in cotton-wool. After the lapse of two or three weeks, electric treatment should be instituted. As faradism generally fails to elicit any response, recourse must be had to an interrupted galvanic current. One pole (cathode) may be placed over an indifferent point, such as the spine, while the other (anode) is slowly stroked over the affected muscles. The weakest current that will cause contraction should be used. The treatment should be given for ten minutes, three or four times weekly, and should be kept up, if necessary, for several months. Massage is a valuable adjuvant to electric treatment. Local bathing with shampooing may also be used with benefit. Internally strychnin ($\frac{1}{100}$ of a grain, gradually increased, to a child of two years) is sometimes useful. The treatment of the latter stages of infantile paralysis is chiefly surgical, and has for its object the prevention or correction of deformities.

CHRONIC ANTERIOR POLIOMYELITIS—PROGRESSIVE MUSCULAR ATROPHY.

(Chronic Spinal Muscular Atrophy of Aran-Duchenne.)

Definition.—A chronic disease characterized anatomically by atrophy of the ganglion-cells in the anterior gray horns of the spinal cord, and manifested clinically by a progressive wasting of the muscles and a corresponding loss of power.

Etiology.—The disease is much more common in males than in females. It occurs most frequently in adults between the ages of twenty and fifty. Heredity is rarely a factor. Exposure, overexertion, mental strain, injury, and syphilis have been mentioned as causes.

Pathology.—Microscopic examination of the cord reveals atrophy or entire absence of the ganglion cells in the anterior cornua and an overgrowth of the neuroglia. The anterior nerve-roots, peripheral motor nerve-fibers, and affected muscles also show degenerative atrophy.

In addition to these lesions, there is often sclerosis of anterolateral white tracts (amyotrophic lateral sclerosis).

Symptoms.—The onset is insidious. The muscles of the hand usually suffer first. The thenar and the hypothenar eminences and the interosseous muscles become more flaccid than normal and gradually waste. Accompanying the atrophy there is a corresponding loss of muscular power. When the interossei no longer afford opposition to the long flexor and extensor muscles, the hand assumes a claw-like position (*main en griffe*), which is quite characteristic. Fine fibrillary tremors or twitchings are almost invariably present in the affected muscles. After the lapse of months, perhaps years, the wasting and paresis spread to the muscles of the shoulder and arm, and then to the neck and trunk. The legs are usually not involved until late, and often escape entirely. Occasionally, however, the disease begins in the lower extremities or back, but this is rare. In the late stages the patient may be reduced to a mere skeleton. Sometimes the process extends to the medulla, in which case the symptoms of *bulbar palsy* are superadded.

There may be some complaint of coldness or of dull pain, but sensation is not impaired. The deep reflexes are lost in the affected limbs and the paralyzed muscles remain flaccid. The reactions of degeneration are sometimes present, but more often there is simply diminished response first to the faradic and then to the galvanic current. The sphincters are not involved.

Diagnosis.— Chronic poliomyelitis must be distinguished from other conditions causing slowly progressing atrophy and weakness, such as amyotrophic lateral sclerosis, muscular dystrophy, multiple neuritis, and syringomyelia.

Amyotrophic Lateral Sclerosis.—In this disease the wasting is associated with spastic rigidity and the tendon-reflexes are exaggerated.

Muscular Dystrophy.—This is commonly an hereditary or a family affection. It occurs in childhood and attacks primarily large muscles (calf, shoulder girth, or face). There is no fibrillary twitching.

Multiple Neuritis.—In this disease the paralysis precedes the wasting. Sensory symptoms are usually prominent, and there is often tenderness along the nerve-trunks.

Syringomyelia.—In this affection the atrophy is accompanied by exaggerated reflexes, peculiar sensory disturbances, and trophic changes in the skin and joints.

Prognosis and Treatment.—The course of the disease is very slow and occasionally marked by remissions. Death may result from involvement of the respiratory muscles, aspiration pneumonia, or bulbar palsy. Treatment is of no avail.

PRIMARY SPASTIC PARAPLEGIA.

(Lateral Sclerosis; Anterolateral Sclerosis.)

Definition.—A chronic disease, characterized by gradual loss of power, marked exaggeration of the reflexes, and a spastic condition of the muscles, without atrophy or sensory disturbances.

Etiology.—The etiology is obscure. The disease usually develops between the ages of twenty and forty. Both sexes are equally affected.

Pathology.—A primary degeneration of the lateral pyramidal tracts (terminations of the upper motor neurons) is assumed to be the anatomic cause of the disease.

Symptoms.—Loss of power is generally the first symptom. This begins in the lower extremities and increases very slowly. The knee-jerk is exaggerated, and in most cases ankle-clonus can be elicited. When put in use, the muscles become stiff or spastic, and when the disease is fully developed, the gait is peculiar. In walking the knees are drawn together, the legs drag behind, and the toes catch the ground.

The muscles do not waste, but tend rather to become hypertrophied from continued reflex stimulation. Sensory and trophic disturbances are absent, and the sphincters are only rarely affected.

Diagnosis.—As an independent affection lateral sclerosis is rare, spastic paralysis of the legs generally being due to (1) *diseases of the brain involving both motor tracts*, as infantile cerebral palsy or (2) *diseases of the spinal cord dividing the lateral columns*, such as multiple sclerosis, tumors of the cord, compression in Pott's disease, transverse myelitis, etc.

Prognosis.—The disease is incurable, but the course is extremely slow.

Treatment.—Rest, warm baths (90° F.), and massage are the most useful measures. If there be a suspicion of syphilis, antiluetic treatment should be instituted.

AMYOTROPHIC LATERAL SCLEROSIS.

Definition.—A chronic disease, characterized anatomically by degeneration of the lateral columns and atrophy of the ganglionic cells in the anterior gray horns of the spinal cord, and clinically by loss of power, atrophy, and a spastic state of the muscles.

Pathology.—The chief lesion is a degeneration of the pyramidal tracts, with atrophy of the large cells in the ventral horns and of certain groups of cells in the medulla.

Symptoms.—These include wasting of the muscles, with loss of power, spastic contractions, and exaggerated reflexes. The upper extremities are usually first affected.

When the medulla is involved, symptoms of glossolabial paralysis appear. Sensation is not impaired, and the sphincters are rarely disturbed.

The muscular rigidity and exaggerated reflexes will distinguish it from pure *progressive muscular atrophy*, and the atrophy of the muscles from pure *lateral sclerosis*.

Prognosis.—Unfavorable. Death occurs in from two to ten years.

Treatment.—This is the same as for lateral sclerosis.

BULBAR PARALYSIS.

(Glossolabiolaryngeal Paralysis.)

Definition.—Paralysis of the lips, tongue, pharynx, and larynx from degeneration of the motor nuclei of the medulla oblongata.

Etiology.—An acute form is observed that results either from hemorrhage or from an acute poliomyelitis of the medulla. The chronic form is essentially a chronic poliomyelitis of the bulb. It may occur as an independent disease, but more often it is a part of amyotrophic lateral sclerosis or progressive muscular atrophy.

Symptoms.—These include impairment of speech; inability to protrude the tongue; dribbling of saliva; difficult swallowing; choking spells from the entrance of food or mucus into the larynx; partial suppression of the voice with measured speaking; and a lack of facial expression. The paresis is attended by atrophy and fibrillary tremors.

In the rare disease known as pseudobulbar paralysis, which results from bilateral lesions in the motor cortex or internal capsule, there is usually some mental impairment and aphasia, and the paralysis is not accompanied by atrophy and fibrillary tremors.

Prognosis.—Unfavorable. The acute variety is speedily fatal; the chronic form may last several years. Death may result from exhaustion, cardiac failure, or aspiration-pneumonia.

Treatment.—This is unsatisfactory. Massage and electricity may be tried. Strychnin has been recommended. The stomach-tube should be used when the patient is unable to swallow.

ACUTE ASCENDING PARALYSIS.

(Landry's Disease.)

Definition.—An acute disease of rare occurrence, characterized by motor paralysis beginning in the feet and rapidly spreading until it involves the muscles of respiration and deglutition.

Etiology.—The causes are unknown. It is usually observed in young male adults. The abrupt onset, acute course, and absence of known cause and of definite lesions have suggested toxic origin.

Pathology.—In a few instances degenerative changes have been detected in the lower motor neurons.

Symptoms.—Febrile symptoms usually usher in the attack. The paralysis begins in the legs and involves successively the trunk, upper extremities, and muscles of respiration and deglutition. The reflexes are abolished. The sphincters are retentive; sensation is usually normal, but there may be some paresthesia; the muscles are relaxed, but do not waste or yield the reactions of degeneration. In some instances the spleen and lymphatic glands are swollen.

Diagnosis.—**Acute Myelitis.**—Anesthesia, wasting, reactions of degeneration, and early involvement of the sphincters will serve to distinguish myelitis from acute ascending paralysis.

Multiple neuritis can usually be distinguished from Landry's disease by the marked sensory disturbances in the former.

Prognosis.—Unfavorable. The vast majority of cases terminate fatally in from a few days to two or three weeks. Very rarely the disease comes to a standstill and a slow recovery ensues.

Treatment.—The patient should be kept at rest, and wet cups applied to the spine. Ergotin (10 to 20 grains a day), belladonna, salicylates, mercury, and iodids are the remedies that have been recommended.

LOCOMOTOR ATAXIA.

(Tabes Dorsalis; Posterior Spinal Sclerosis.)

Definition.—A degenerative affection of the posterior columns of the spinal cord and posterior nerve-roots, characterized by incoördination, loss of deep reflexes, disturbances of sensation and nutrition, and various ocular phenomena.

Etiology.—The disease occurs most frequently between the ages of thirty and fifty. It is ten times more common in men than in women. Syphilis appears to be the exciting cause of at least three-fourths of all cases. Exposure, excesses, overexertion, and alcoholism are contributing factors.

Pathology.—The pia mater over the posterior columns is somewhat thickened and opaque. The posterior columns have a grayish color and are firm and shrunken.

Microscopic examination reveals atrophy of the nerve-fibers and overgrowth of neuralgia in the columns of Goll and Burdach. The posterior nerve-roots are invariably degenerated. The spinal ganglia may or may not be involved. In many cases the sensory nerves of the periphery and the cranial nerves, especially the optic, exhibit degenerative changes.

Symptoms.—*Motor Phenomena.*—One of the earliest symptoms is loss of coördination. This is first manifested by unsteadiness when the patient walks in the dark. When he stands erect, with the eyes closed and feet together, he staggers and tends to fall (Romberg's symptom). When the arms are affected, there is inability to perform work requiring delicate coördination, such as writing and piano-playing. This loss of coördination in the upper extremities becomes conspicuous when the patient, while his eyes are closed, attempts to touch the tip of his nose.

The gait is characteristic; in walking he raises his feet high, throws them forward, and brings them down forcibly in such a way that the whole sole strikes the floor at once. Although the patient may be unable to walk or to use his hands with precision, there is very little loss of power. A peculiar relaxation of the muscles with an unusual mobility of the joints (hypotonia) is not uncommon.

Sensory Phenomena.—Pain is rarely absent; it is sharp and lancinating in character, and appears in paroxysms. It usually involves the extremities, but sometimes it attacks the stomach and is accompanied with obstinate vomiting. The term *gastric crisis* is applied to this phenomenon. Crises may also occur in other organs, notably the larynx, where they are manifested by intense dyspnea and stridulous breathing.

A sense of constriction about the trunk at different levels ("girdle sensation") is a common sensory symptom. Various forms of paresthesia are observed, such as tingling, numbness, burning, etc. Irregular areas of anesthesia are nearly always present. The muscle-sense is also more or less impaired.

Reflex Phenomena.—The knee-jerk is lost early in the disease. Later other reflexes, such as the plantar, cremasteric, and abdominal, may be abolished.

Eye Phenomena.—The pupil fails to respond to light while it still accommodates for distance (Argyll-Robertson pupil). The pupils are usually small. Optic-nerve atrophy and paresis of the ocular muscles are frequent symptoms.

Visceral Phenomena.—Apart from the crises already mentioned, there may be incontinence of urine, constipation, or paralysis of the sphincter ani, and loss of sexual power.

Trophic Phenomena.—These usually appear late. The most curious are the so-called arthropathies, which consist of enlargement of the joints, associated with serous effusions, atrophy of the heads of the bone, erosion of the cartilages, and calcification of the ligaments. These articular changes sometimes lead to luxations. Occasionally a perforating ulcer appears in the foot.

Mental Phenomena.—In many cases symptoms of paretic dementia ultimately supervene.

Diagnosis.—**Multiple Neuritis.**—In this disease the onset is rapid, eye-symptoms and lightning pains are absent, the bladder is rarely affected; while, on the other hand, there is an actual loss of muscular power, with wasting of the muscles and diminished electric excitability.

Cerebellar Disease.—In lesions of the cerebellum the knee-jerks are usually retained, the pupils react to light, lightning pains and other sensory disturbances are wanting, while headache, vertigo, optic neuritis, and vomiting are prominent symptoms.

Gastralgia.—A gastric crisis may be mistaken for gastralgia, but the associated phenomena of locomotor ataxia will prevent an error in diagnosis.

Prognosis.—Complete recovery probably never occurs. The duration ranges from three to twenty years. Death is usually the result of some intercurrent disease.

Treatment.—Rest is an important factor in the treatment. Erb advises that the patient should live as if he were an old man, quietly, regularly, and with no excitement. Mental fatigue should also be avoided. Sexual excesses are exceedingly injurious. The diet should be nutritious and easily digestible. Alcohol and tobacco should be used sparingly, if at all. Flannel should always be worn next to the skin.

Massage affords a valuable means of securing the benefits of exercise without the expenditure of energy.

Systematic reëducation of coördinating movements, as originally recommended by Frenkel, has been found a most effective remedy for the ataxia. Even in advanced cases, in which there is marked disturbance of sensation, this method of treatment is not without benefit, and the improvement may last for years if the disease is stationary or only slowly progressive.

Tepid baths of 80°–85° F. are sometimes of distinct service. They should be suspended, however, while the exercise treatment is being used. Mercury and iodids should be given a thorough trial in all cases in which syphilis is suspected.

The Pains.—When the pains are severe, the most potent remedial measure is absolute rest in bed. Light touches of the actual cautery or sinapisms over the root of the nerve supplying the affected part often afford relief. Deep massage is sometimes of service. Mitchell has found the alternate application of ice and hot water useful. Flannel bandages applied firmly from the toes up to the middle third of the thigh sometimes do much good. A snugly fitting abdominal binder may also be used to lessen girdle pain. Electricity in the form of the faradic brush, static spark, or stabile galvanic anode is worthy of a trial.

The most generally useful anodynes are phenacetin and antipyrin. Cannabis indica or nitroglycerin occasionally succeeds.

In many cases recourse must be had to morphin, but its use should be deferred as long as possible.

Numbness and *paresthesia* often yield for a time to local applications of faradism given with the wire brush.

Vesical weakness should receive the most careful attention. The bladder must be thoroughly emptied—if need be, by catheterization. On the first appearance of cystitis the bladder should be thoroughly washed out with weak antiseptic solutions.

ATAXIC PARAPLEGIA.

Definition.—A sclerotic affection of the posterior and lateral columns, manifesting symptoms of both locomotor ataxia and spastic paraplegia.

Symptoms.—It resembles spastic paraplegia in the loss of power, spastic condition of the muscles, increased reflexes, and absence of ocular and sensory disturbances; and locomotor ataxia in the distinct loss of coördination.

DISSEMINATED CEREBROSPINAL SCLEROSIS.

(**Multiple Sclerosis; Insular Sclerosis.**)

Definition.—A chronic disease, characterized anatomically by patches of sclerosis of varying size scattered throughout the brain and spinal cord.

Ætiology.—The causes that lead to other scleroses of the spinal cord may induce this disease; the infectious fevers, however, are assigned a prominent place in its etiology. It is a disease of youth, the majority of cases occurring between the tenth and thirtieth years.

Pathology.—Areas of firm, gray, sclerotic tissue, of various sizes and shapes, are found through the brain and spinal cord.

Symptoms.—The symptoms develop slowly and are variable. The most characteristic are: (1) A coarse tremor, which affects chiefly the arms and head, which is brought on by muscular effort, and which ceases during complete rest (volitional or intention tremor); (2) nystagmus, or oscillation of the eye-balls; (3) slow, jerky, scanning speech; (4) weakness of the legs, with rigidity of the muscles and increased knee-jerks. In addition there may be vertigo, mental impairment, optic atrophy, incontinence of urine, and epileptiform or apoplectiform seizures.

Paresthesia is sometimes present, but anesthesia is rare. Trophic disturbances are very uncommon. The course is long, from 2 to 20 years, and remissions not rarely occur.

Diagnosis.—**Paralysis Agitans.**—In this disease the mask-like face, attitude, and gait are characteristic. The tremor decreases during effort and persists during repose; nystagmus and scanning speech are absent. *Hysteria* may simulate multiple sclerosis, but it does not present nystagmus, optic atrophy, or bladder disturbances.

Treatment.—The general treatment is the same as that of posterior sclerosis. Bromids, hyoscin, hyoscyamin, and belladonna have been recommended for the tremors.

HEREDITARY ATAXIA.

(Friedreich's Ataxia.)

Definition.—A family disease, characterized anatomically by sclerosis of the lateral and posterior columns of the spinal cord, and clinically by symptoms resembling those of locomotor ataxia.

Etiology.—The disease most frequently develops between the second and fifteenth years, and usually attacks a number of individuals in the same family. Some cases can be traced to heredity; in others no cause can be ascertained.

Pathology.—The chief lesion is a sclerosis of the lateral and posterior columns of the spinal cord.

Symptoms.—The characteristic symptoms are: Ataxia of all purposeful movements; loss of the knee-jerks; irregular jerking movements of the hands; muscular weakness; nystagmus; a scanning speech; lateral spinal curvature, and deformity of the feet, generally talipes equinus with extension of the big toe.

It differs from locomotor ataxia in the absence of sharp pains, of anesthesia, of the Argyll-Robertson pupil, and in the occurrence of nystagmus, scanning speech, and deformities.

Prognosis and Treatment.—Unfavorable. The course is extremely slow. Treatment is of no avail.

SYRINGOMYELIA.

Definition.—A chronic affection of the spinal cord, characterized anatomically by the formation of a cavity in its substance, and clinically by atrophy of certain muscles, peculiar disturbances of sensation, and various trophic disorders.

Etiology.—It is much more common in males than in females. Eighty per cent. of the cases occur between the ages of ten and forty years. Traumatism or one of the infectious fevers may excite it.

Pathology.—The disease begins as an overgrowth of the embryonic neuroglia. The cavity-formation is a secondary process, and is brought about by degeneration of the gliomatous tissue, or possibly in some instances by hemorrhage. The cervical and upper dorsal regions are the usual seats of the lesion. The cavity lies in the gray matter, and may be in the position of the central canal or somewhat posterior to it. Secondary degenerations are frequently observed in the anterior or posterior horns or in the anterior or posterior columns.

Symptoms.—The disease usually attacks the upper extremities, the chief symptoms being wasting of the muscles; fibrillary tremors; loss of painful and thermic sensations, while tactile sensation is preserved or but slightly affected (*dissociation symptom*); lateral spinal curvature; and various trophic disturbances, such as arthropathies, fissures, ulcers, and gangrene. Such eye symptoms as nystagmus, inequality of pupils, and narrowing of the visual fields are frequently observed. In many instances symptoms of lateral sclerosis, posterior sclerosis, or bulbar disease are superadded.

The distinctive features of *Morvan's disease* (probably a form of syringomyelia) are tactile anesthesia and painless felons.

Diagnosis.—**Cervical pachymeningitis** is more painful, and the anesthesia includes tactile sensation. In **progressive muscular atrophy** and **amyotrophic lateral sclerosis** sensory symptoms are wanting. **Leprosy** may be recognized by loss of tactile sensation, discoloration of skin, nodular swellings, and presence of bacilli in the secretions of the nose and eyes and in the serum of blisters.

Prognosis.—Unfavorable. The duration is from five to twenty years.

Treatment.—This is necessarily symptomatic.

CAISSON DISEASE.

(**Divers' Paralysis.**)

Definition.—A condition observed in divers and others subjected to increased atmospheric pressure, and characterized by motor and sensory paralysis and other nervous symptoms.

Etiology.—A pressure of more than two atmospheres is required to produce the paralysis, and the time elapsing before its appearance lessens as the pressure increases.

Pathology.—The symptoms have been ascribed by some to the liberation in the cord of gases that have been absorbed by the blood during exposure to the high pressure; by others, to stasis of blood and edema. The cord is found congested and sometimes the seat of hemorrhages.

Symptoms.—The condition may manifest itself immediately on reaching the surface or after the lapse of several hours. The most important phenomena are pains in the joints, followed by motor and sensory paralysis in the lower extremities. The bladder and rectum are sometimes involved. Occasionally the paralysis takes the form of a hemiplegia instead of a paraplegia. Gastralgia and vomiting are common symptoms. In severe cases coma develops and death follows in a few hours. Generally, however, the symptoms gradually subside, and the power is fully restored in the course of a few days or a few weeks.

Treatment.—As a preventive measure, the transition from high to low pressure should be accomplished gradually. Marked cases should be treated as acute myelitis.

DISEASES OF THE NERVES.

NEURITIS.

Definition.—Inflammation of nerves.

Etiology.—(1) It may result from traumatism—blows, wounds, or compression. (2) It may be due to exposure to cold and wet. (3) It may be secondary to inflammation of adjacent structures. (4) It may be secondary to rheumatism, gout, syphilis, or one of the infectious fevers.

Pathology.—The sheath, interstitial connective tissue, or fibers may be independently affected, but, as a rule, all parts of the nerve are involved. When the process is acute, the nerve is red and swollen, and microscopic examination reveals an infiltration of leukocytes, with more or less granular degeneration of the fibers.

In *chronic neuritis* the nerve-trunk is gray, shriveled, and hard, and microscopic examination shows an overgrowth of connective tissue and granular degeneration of fibers.

Symptoms of Acute Neuritis.—There are three sets of phenomena—sensory, motor, and trophic.

Sensory Symptoms.—There is severe pain following the course of the affected nerve, which is tender to the touch. The pain is often associated with various manifestations of paresthesia, such as burning, numbness, tingling, and the like. The part is at first hyperesthetic, but later it is more or less anesthetic.

Motor Symptoms.—Muscular power is impaired; there may be fibrillar tremors; the reflexes are diminished or lost.

Trophic Symptoms.—An eruption of herpes sometimes follows the affected nerves. The skin may become glossy and the nails lusterless and brittle. In advanced cases there are wasting of muscles and impaired electrocontractility. Occasionally effusion into the joints is observed.

In severe cases there may be febrile symptoms.

Chronic neuritis is characterized by pain, anesthesia, paresis, atrophy and contracture of the muscles, reactions of degeneration, "glossy skin," and thickening and brittleness of the nails.

Diagnosis.—Neuritis may be mistaken for **neuralgia**; but in the latter the pain is paroxysmal and is unassociated with tenderness along the course of the nerve, paresthesia, anesthesia, paresis, and changes in the electrocontractility.

Prognosis.—In acute cases the prognosis is guardedly favorable; the duration is from a few days to several weeks. In chronic neuritis, after the development of marked trophic changes, the prognosis is grave.

Treatment.—The cause should be ascertained, and, if possible, removed. In rheumatism, alkalis and salicylates are indicated. In syphilis, iodid of potassium should be administered in large doses. The part should be put at rest. For the pain, sedative lotions (lead-water and laudanum), warm fomentations, or small blisters may be applied to the affected parts, and morphin administered hypodermically. When morphin is contraindicated, salicylic compounds or phenacetin may be employed in its stead. Such a combination as the following is often efficacious:

℞.	Acetanilidi	ʒj
	Salophen	ʒiss
	Codeinæ sulphatis	gr. ij.—M.

Fiant chartulæ No. xij.
SIG.—One every three or four hours.

After the acute symptoms have subsided massage and electricity should be resorted to, in order to restore the functions of the nerve.

MULTIPLE NEURITIS.

Definition.—Inflammation of several nerve-trunks resulting from a general cause, and characterized by pain, paresthesia, anesthesia, paresis, and muscular atrophy.

Etiology.—The disease may result from (1) exposure to cold and wet; (2) specific infections, such as diphtheria, influenza, measles, rheumatism, etc.; (3) certain poisons derived from without, such as alcohol, lead, arsenic, carbon monoxid, sulphonal, etc.; (4) certain auto-intoxications, such as occur in gout and diabetes; (5) malnutrition, as in advanced arteriosclerosis. In the Orient multiple neuritis occurs as an endemic disease (*beri-beri* or *kakke*) which is probably of microbic origin.

Symptoms.—The **acute form** is characterized by chilliness, moderate fever (102°–103° F.), pains in the head and back, anorexia, constipation, and the following local phenomena: pain, numbness, and tingling in the affected limbs, loss of power, especially in the legs and extensor muscles of the wrist, tenderness over the nerve-trunks, abolition of reflexes, and more or less anesthesia.

Death may occur within one or two weeks from cardiac or respiratory paralysis. Generally, however, recovery follows in from a few weeks to several months.

Chronic Form.—*Sensory symptoms* are prominent. Numbness, tingling, hyperesthesia, and intermittent pains appear early and are followed by some anesthesia, especially of the legs and hands. The nerve-trunks are sensitive.

Motor Symptoms.—Weakness of the legs and forearms develops rapidly. The typical paresis is foot-drop and wrist-drop. The sphincters are not affected. The knee-jerks are lost. As a rule the paralyzed muscles do not respond to the faradic current, but yield the reaction of degeneration with the galvanic current. If the patient is able to walk, his gait is characteristic. To avoid dragging his toes, he raises the foot high, throws it suddenly forward, and brings it down flat on the floor, as if walking over obstacles (steppage gait).

Vasomotor and Trophic Disturbances.—The paralyzed muscles are flabby and soon waste. Edema of the feet and hands, and local sweating are often seen. Changes in the nails are common. Bedsores do not appear.

Mental Symptoms.—Delirium with hallucinations and illusions are frequently observed in the alcoholic type.

Diagnosis.—*Locomotor Ataxia.*—The absence of the lightning-pains, girdle sensation, Argyll-Robertson pupil, and the presence of paralysis, wasting, and neural tenderness will serve to distinguish multiple neuritis from locomotor ataxia.

Prognosis.—Recovery usually occurs in time, when the cause can be removed.

Treatment.—This is the same as for localized neuritis.

SCIATICA.

Definition.—Pain along the sciatic nerve, usually resulting from neuritis.

Etiology.—It is usually primary, developing in rheumatic or gouty persons after exposure to cold and wet. Some cases owe their origin to syphilis. Occasionally it is a secondary condition resulting from the presence of an intrapelvic growth or from caries of the bone in hip-joint disease.

Symptoms.—The disease may begin abruptly or gradually, and is characterized by a sharp shooting pain running down the back of the thigh. Movement of the limb intensifies the suffering. The pain may be uniformly distributed along the course of the nerve, but not infrequently there are certain spots where it is more intense. Subjective sensations, such as tingling and numbness, are often noted. The nerve may be extremely sensitive to touch. The symptoms grow worse at night and on the approach of stormy weather. The duration of the attack varies from a few days to several months. In long-standing cases the muscles become atrophied and rigid.

Diagnosis.—**Coxalgia.**—In this affection the pain is most marked in the hip- and knee-joints; pressure over the trochanter elicits pain; and the nerve is not tender to the touch.

Prognosis.—Recovery follows in the majority of cases when treatment is instituted early and is persistently carried out. In some cases relapses occur frequently, and finally the pain becomes more or less continuous.

Treatment.—The first indication is to remove the cause. In acute cases rest in bed is essential. In severe cases the limb should be immobilized by means of salt bags or a long straight splint. Free evacuation of the bowels should be secured in order to deplete the pelvic veins. When there is a history of rheumatism or the attack has been induced by cold, salicylates should be given in full doses. When there is reason to suspect syphilis, iodids should be given a fair trial. Irrespective of the cause, phenacetin or antipyrin may be useful in relieving pain. Counterirritation often affords much relief. When the pain is very severe, this is best accomplished by means of small blisters or light touches of the actual cautery applied over the points of greatest tenderness. In some cases acupuncture acts very satisfactorily. In milder cases the Scottish douche—in which a stream of warm water of gradually increasing temperature is directed on the course of the nerve until the pain subsides, when it is suddenly changed for a cold jet—is an efficient remedy. Agonizing pain must be relieved by injections of cocain ($\frac{1}{4}$ grain), chloroform (5 to 10 minims), guaiacol (2 to 3 minims), or morphin ($\frac{1}{8}$ to $\frac{1}{4}$ grain), made deeply and as near to the nerve as possible. Morphin should be withheld as long as possible. In some cases deep injections of distilled water act remarkably well. Massage is indicated only when the acute symptoms have subsided, and should then not be too energetic.

FACIAL PARALYSIS.

(Bell's Palsy.)

Etiology.—Paralysis of one side of the face may result: (1) From a tumor, clot, or abscess involving the facial center on the cortex of the brain or the nucleus of the facial nerve; (2) from the pressure of inflammatory exudate on the nerve-trunk between the brain and the skull; (3) from paralysis of the nerve within the petrous portion of the temporal bone, excited by a fracture or by an extension of inflammation of the middle ear; (4) from inflammation of the peripheral filaments, excited by exposure, injury, rheumatism, or one of the infectious fevers.

Symptoms.—The side affected is expressionless; the natural lines are obliterated; the angle of the mouth droops; the eye cannot be closed; tears flow over the cheek; and speech is affected from an inability to pronounce the labials. When the patient attempts to laugh or whistle, the absence of movement on the affected side becomes still more conspicuous. In peripheral neuritis the reflexes are abolished; and when the nerve is involved in the temporal bone, there may be a loss of taste in the anterior part of the tongue.

Diagnosis.—When the lesion is supranuclear the upper muscles of the face (orbicularis palpebrarum and frontalis) usually escape, voluntary movements are more impaired than emotional movements, electric reactions are normal, and there is generally hemiplegia.

When the lesion is nuclear or infranuclear all the muscles of one side of the face, including those of the forehead and eye, are involved, both emotional and voluntary movements are lost, and the electric reactions are altered in character. In nuclear lesions other cranial nerves are usually involved with the facial. In pontine lesions there is often paralysis of the limbs on the side opposite to the facial palsy (crossed paralysis). When the nerve is involved within the Fallopian canal there is frequently loss of taste in the anterior part of the tongue on the paralyzed side.

Prognosis.—The prognosis will vary with the cause. It should be guardedly favorable when the paralysis is due to peripheral neuritis.

Treatment.—The cause should be ascertained, and, if possible, removed. In paralysis of centric origin little can be done except in syphilitic cases. In middle-ear disease remedies should be directed to that origin. When paralysis results from inflammation of the peripheral filaments of the facial nerve, blisters should be applied near the stylomastoid foramen. Later, a course of iodid of potassium will be useful, and restoration of power may be materially assisted by massage and electricity.

FUNCTIONAL NERVOUS DISEASES.

HEADACHE.

(Cephalalgia.)

Headache of Organic Brain Disease.—This form is observed in meningitis, cerebral tumor, abscess, softening, etc., and may be recognized by its persistence and by the associated evidences of organic cerebral disease, such as optic neuritis, mental aberration, paralysis, especially of the facial muscles, and vomiting arising independently of other gastric symptoms.

Under this head is included the headache of *syphilis*, which may be diagnosed by the history; by the other evidences of syphilis; by its frequent association with somnolence; and by the effect of iodid of potassium.

Headache of Cerebral Hyperemia.—*Active cerebral congestion* usually results from prolonged mental work, fever, or exposure to the sun. Toxic and reflex headaches are often directly due to active cerebral congestion, but these will be discussed later.

Passive cerebral congestion may result from obstruction to the return of blood from the brain, as by a tumor of the neck or cardiac disease. It is also common in elderly people from a relaxed condition of the vessels.

In cerebral congestion the headache is of a throbbing or bursting character; the head is hot; the face flushed; the eye-ground injected; and the distress is increased by lowering the head.

The exciting cause must be determined by the history and by a careful examination of the various organs, especially the heart.

Headache of Cerebral Anemia.—This is frequently dependent upon general anemia. It is also common in neurasthenia resulting from overwork, prolonged emotional excitement, excesses, etc. More rarely it is dependent upon aortic stenosis.

In cerebral anemia the pain is frequently vertical; it is not throbbing, but it is described as a sensation of weight or gnawing; the extremities are cold; the face and eye-grounds are pale; the mind is depressed; fainting spells are often present; lowering the head and the inhalation of nitrite of amyl relieve the pain.

Reflex Headache.—Headache is often due to *eye-strain* resulting from refraction errors, and in obstinate cases a careful examination of the eyes should always be made. Headache of this origin is frequently a browache, and may be associated with restlessness, vomiting, and insomnia. It is induced or aggravated by prolonged use of the eyes.

Ovarian or uterine diseases often produce a reflex headache. It is usually located at the vertex, and is relieved by pressure of the hand.

Gastric irritation is responsible for many headaches; the latter are invariably relieved by vomiting, and are usually associated with other evidences of stomachic disorder.

Nasal catarrh may induce persistent headache, which is generally confined to the forehead, temples, or vertex, and is aggravated by exacerbations of the catarrh. The pain is often associated with tenderness of the inner wall of the orbit, and is increased by irritating the nasal mucous membrane with a probe.

Toxemic Headache.—A persistent headache often results from Bright's disease, and is *uremic* in origin. It may be recognized by the high arterial tension and by the albumin and casts in the urine. A urinary analysis should be made in all cases of persistent headache.

Gout produces an intractable headache that is associated with vertigo, great irritability of temper, and a "brick-dust" deposit in the urine.

Chronic malarial poisoning may manifest itself in a headache which is usually confined to the supra-orbital region. It is apt to recur at regular intervals, is often associated with tenderness over the supra-orbital nerve, and is relieved only by large doses of quinin.

A headache of *rheumatic* origin sometimes develops in those subject to rheumatism. It is frequently excited by exposure or a sudden change of temperature. It usually affects the aponeurosis of the occipitofrontalis and temporal muscles, is increased by wrinkling the forehead and forcibly moving the jaws, and is associated with tenderness of the scalp.

Alcoholism is often associated with headache. In acute alcoholism the headache probably results from cerebral hyperemia; in chronic alcoholism it is often due to a low grade of meningitis.

Among other headaches of toxic origin may be mentioned those due to constipation, lead-poisoning, diabetes, infectious fevers, and absorption of foul gases.

Hysteric Headache.—In hysteria there is often a persistent headache, which grows worse at the menstrual periods, and which improves under pleasurable excitement. It may be diffuse, but frequently it is localized, and is described as resembling the effect that would be produced by a nail being driven into the head; hence it has been termed *clavus*.

Diagnosis.—Headache must be distinguished from *migraine*. In the latter the attacks are usually more distinctly periodic; the pain is often unilateral, and is frequently accompanied by vomiting, vasomotor disturbances, and subjective visual phenomena.

Headache in the region of the orbit may be mistaken for *acute glaucoma*, but in the latter condition the eye is inflamed; the cornea is hazy; the pupil is sluggish; vision is impaired; and on palpation the affected eyeball is found to be harder than its fellow.

Treatment.—In the interval between the attacks careful search should be made for the cause, which, if possible, must be removed. In the reflex headache of eye-strain, the adjustment of proper glasses is often all that is required.

In the headache of gastric origin appropriate remedies should be directed to the stomach. In the headache of anemia a nutritious diet, with iron, arsenic, and other tonics, will be required. In headaches of uremic origin a milk diet with measures calculated to increase the action of the skin, bowels, and kidneys will often afford considerable relief. In malarial headache quinin in large doses with arsenic will effect a cure.

The Attack.—In headache dependent upon gastric acidity, after unloading the stomach with a non-irritating emetic, bromid with antacids will prove useful, thus:

℞. Sodii bromidi . . . . ʒij
  Spiritus ammoniæ aromatici . . . . . f℥ij
  Aquæ . . . . q. s. ad f℥iij.—M.
SIG.—A tablespoonful every hour or two.

In headache of acute cerebral congestion the feet should be soaked for ten or fifteen minutes in very hot water; an ice-bag placed on the head; and some sedative like the following administered:

℞. Acetphenetidini . . . . ʒj
  Sodii bromidi . . . . ℥ss.—M.
Fiant chartulæ No. xij.
SIG.—One powder every hour or two until relieved.

When the attack is very severe, aconite (1 or 2 drops) may be given every hour or two.

In cerebral anemia relief temporarily follows the use of antipyrin or phenacetin, especially in combination with caffein, thus:

℞. Acetphenetidini . . . . ʒiss
  Caffeinæ citratæ . . . . gr. xxiv.—M.
Fiant chartulæ No. xij.
SIG.—One as required.

In rheumatic headache salicylic compounds are very useful; they may be combined with phenacetin or antipyrin:

℞. Acetphenetidini
  Salophen . . . . āā ʒiss.—M.
Fiant chartulæ No. xij.
SIG.—One every two or three hours.

In uremic headache the diet should be restricted to milk, action of the bowels secured by a saline draft, and diuresis encouraged by digitalis, caffein, or the vegetable salts of potassium:

℞.	Potassii citratis	ʒij
	Spiritus juniperi	f℥vj
	Spiritus ætheris nitrosi	f℥ij
	Infusi scoparii	f℥vj.—M.

SIG.—A wineglassful thrice daily. (DAY.)

VERTIGO.

(Dizziness; Giddiness; Swimming in the Head.)

Definition.—A sense of unstable equilibrium in which the patient himself or surrounding objects appear to be in a state of rapid oscillation or rotation. It is a symptom of many conditions.

Etiology.—Vertigo may result from:

1. Cerebral anemia or congestion. The dizziness preceding a fainting fit is an illustration of the former, and that following exposure to the rays of the sun is an illustration of the latter. Vertigo is often a pronounced symptom of chronic heart disease and of arteriosclerosis is included under this head.

2. Diseases of the ear. Vertigo may occur in any affection of the ear, but it is especially severe in the symptom-complex known as Ménière's disease (see p. 434).

3. Palsy of the ocular muscles. This form of vertigo is often associated with nystagmus and is relieved by closing the eyes.

4. Reflex irritation. The most common example of this form is the vertigo dependent upon gastric disturbances. It is also noted in some cases of nasal obstruction and ovarian disease.

5. Organic disease of the brain and cord. Cerebral tumor, meningitis, and softening are frequently associated with vertigo. It is often quite marked in cerebellar disease. It may be a pronounced symptom in disseminated sclerosis and locomotor ataxia.

6. Toxic substances in the blood. The vertigo observed in gout, uremia, and diabetes is included under this head.

When taken in large doses, certain drugs, as alcohol, belladonna, cannabis indica, lobelia, and conium, may produce the symptom. It is often a marked symptom of chronic lead-poisoning.

7. Epilepsy. Vertigo may precede, follow, or take the place of an epileptic seizure.

8. Psychic disturbances. Vertigo is not uncommon in hysteria, neurasthenia, and the traumatic neuroses.

9. Unknown causes. The term *essential vertigo* has been applied to those cases in which, after the most exhaustive study, no adequate cause can be ascertained. There is sometimes a hereditary tendency to this form of vertigo.

Diagnosis.—Vertigo must be distinguished from **petit mal,** or **minor epilepsy.** The history, the presence of a definite cause, and the absence of unconsciousness and of convulsive movements will serve to separate vertigo from epilepsy.

The determination of the cause of the vertigo must be based upon the history, the age at which it develops, and a critical examination of the various organs.

Prognosis.—This will depend entirely on the cause; when the latter can be removed, the prognosis is favorable.

Treatment.—This must be directed to the causal condition.

MÉNIÈRE'S DISEASE.

(Labyrinthine Vertigo; Aural Vertigo.)

Definition.—Paroxysmal vertigo, probably depending upon disease of the internal ear.

Ætiology and Pathology.—The exact cause of Ménière's disease is still undetermined. In some cases, however, inflammatory changes have been observed in the semicircular canals. Very severe acute attacks are sometimes observed in patients previously healthy. In these the lesions are probably an active hyperemia of, or a hemorrhage into, the labyrinth. It is probable that mild forms of the disease can be indirectly induced by lesions of the middle ear.

Symptoms.—Frequently prodromes precede the attack, such as deafness or earache. These, however, may be absent, and the attacks ushered in with extreme vertigo and tinnitus aurium. The latter is often compared to the escape of steam, the buzz of an insect, or the discharge of a cannon. The patient feels as if he or surrounding objects were being whirled violently around, and in severe cases the face is pale and anxious; the surface is clammy; there are nausea and vomiting; and the patient falls unconscious.

As a rule, there is deafness in one ear at least, but exceptionally hearing may be quite normal. At first the paroxysms may occur at long intervals, but as the disease advances they become more frequent and the tinnitus and deafness become more marked.

Diagnosis.—The paroxysmal vertigo, deafness, and tinnitus aurium are the diagnostic features.

Prognosis.—The prognosis should always be guarded. Some cases recover entirely, but in the majority the vertiginous attacks continue until the deafness in the affected ear becomes complete.

Treatment.—The middle ear should be carefully examined, and any existing disease treated. Severe counterirritation by blisters or the actual cautery applied behind the ear may be of some service. Bromid of potassium, hydrobromic acid, cimicifuga, or gelsemium in full doses sometimes afford temporary relief. Daily injections of pilocarpin ($\frac{1}{6}$ grain), as originally recommended by Politzer, are occasionally of service.

EPILEPSY.

(Idiopathic Epilepsy; Falling Sickness.)

Definition.—A chronic disease of the nervous system, characterized by periodic atacks of unconsciousness, which may or may not be associated with convulsive seizures.

Etiology.—Heredity predisposes, and the ancestral disease may not have been epilepsy, but insanity, hysteria, or another neurosis. It generally begins before puberty, and very rarely after the twenty-fifth year. All causes that impair the health and exhaust the nervous system exert a predisposing influence. The reflex convulsions of children resulting from gastric irritation, worms, etc., if long continued, may induce chronic epilepsy. In these cases, although the exciting cause has been removed, the habit of spontaneous motor discharge, through constant repetition, is established, and may continue through life. In those subject to convulsions, overwork, gastric irritation, or excitement may precipitate an attack.

Pathology.—No demonstrable causal lesions are detected. The disease apparently depends upon an instability of the motor centers, so that from trivial exciting causes violent discharges occur from time to time.

Symptoms.—*Grand Mal.*—The seizure is often preceded by a peculiar sensation termed an aura, beginning in a finger or toe, and rising until it involves the head, when the patient gives a shrill scream and falls to the floor unconscious. At first the face is pale, the pupils contracted, and the body thrown into a tonic spasm in which the head is retracted and rotated, the limbs forcibly extended, and the thumbs turned into the palms and firmly clenched by the flexed fingers. In a few seconds the tonic spasm relaxes, the movements become clonic or intermittent, the pupils dilated, and the face cyanosed. From the violent contraction of the masseters frothy saliva, often blood-streaked, pours from the mouth. The clonic spasms continue for a minute or two, and are generally followed by a period of coma lasting from a few minutes to several hours. Sometimes the patient returns at once to consciousness, and complains simply of weakness, muscular soreness, and mental confusion. More rarely the convulsion is followed by an outbreak of mania or of *epileptic automatism*, a condition in which the patient unconsciously performs simple or complicated acts.

Petit Mal.—In this type the seizure consists of momentary unconsciousness, with pallor, and rarely twitching of the muscles. The patient suddenly stops in the midst of his work or conversation, remains quiet for a few seconds, and then continues where he left off, perhaps unconscious of the interruption. *Petit mal* may be a forerunner of *grand mal* or may alternate with it.

Between these two extremes the seizures manifest all grades of severity. The frequency of the paroxysms varies considerably: they may occur as seldom as once a year or as often as ten or twelve times a day. A marked periodicity in their recurrence is often observed.

The term "status epilepticus" is applied to a condition in which the convulsions follow each other in rapid succession without a return of consciousness.

The epileptic may manifest no other symptoms beyond the convulsions, but when the latter are very frequent, the health fails and the mental power deteriorates.

Diagnosis.—The convulsions of **chronic organic disease of the brain** may be mistaken for those of idiopathic epilepsy, but the former do not often develop before the age of twenty-five; they are frequently confined to one member or begin in one member and then become general (Jacksonian epilepsy); and they may be associated with a history or the concomitant symptoms of syphilis, or with evidences of local injury.

Uremia.—Uremic convulsions may be recognized by the history and the results of the urinary analysis.

Prognosis.—Generally unfavorable. Arrest of the disease is rare, but amelioration is often secured by treatment.

Treatment.—Hygienic treatment is of the utmost importance. Moderate exercise, both mental and physical, is beneficial. Idleness and seclusion have a baneful effect. Home training must be carried on with the greatest care, much tact and firmness being required to prevent loss of self-control. Children who are particularly irascible are often much better trained in a special institution. As a rule, a diet that is for the most part vegetable will be found to be best adapted to the patient's condition, but when the disease is associated with lowered vitality, a fair amount of animal food should be permitted. Overloading the stomach is a potent factor in precipitating attacks. The bowels must be regulated by diet, and, if necessary, by mild aperients. Liberal water-drinking, frequent bathing, followed by friction of the skin, light exercise in the open air, and other measures that favor elimination are to be recommended. General tonics, like iron, arsenic, and cod-liver oil, are sometimes required to combat anemia and malnutrition.

Although very few cases of epilepsy are purely reflex, local irritation—phimosis, adherent prepuce, worms, a foreign body in the nose or ear, and painful cicatrices—should be carefully sought for and, if found, removed.

The most reliable drugs are the bromids: one or two drams of a combination of the bromids of sodium, potassium, and ammonium may be given daily. Strontium bromid is often efficacious, and it is less depressing than the other bromids. The tendency to acne may be considerably lessened by the addition of a drop or two of Fowler's solution with each dose. A small amount of antipyrin often lessens the amount of the bromid required to check the convulsions.

℞. Potassii bromidi
Ammonii bromidi . . . . āā ʒiij
Liquoris potassii arsenitis . . . . . . fʒj
Antipyrinæ . . . . ℥j
Aquæ menthæ piperitæ . . q. s. ad f℥vj.—M.
SIG.—A tablespoonful in water night and morning.

In nocturnal epilepsy chloretone (5 grains) is often a useful adjuvant to the bromids. Horse-nettle (Solanum carolinense) is another remedy that appears to increase the activity of the bromids. From ½ to 1 fluidram of the fluid extract may be given thrice daily. In petit mal nitroglycerin ($\frac{1}{100}$ to $\frac{1}{20}$ grain) is sometimes efficacious.

Trephining offers some hope of relief in certain cases of focal epilepsy, although it has to its credit less than 4 per cent. of recoveries.

The Attack.—When an aura is perceived, it is often possible to arrest the paroxysm by the inhalation of amyl nitrite. Patients may provide themselves with this drug in the form of pearls that may be crushed in the handkerchief. If a sensory aura is felt in a limb, the part may be firmly grasped or encircled with a light ligature. During the attack the head should be slightly raised, the clothes loosened, and a piece of cork or firm rubber pushed between the teeth. In the status epilepticus the most reliable measures are inhalations of chloroform, ether, or amyl nitrite, hypodermic injections of hyoscin ($\frac{1}{100}$ grain) or of morphin ($\frac{1}{4}$ grain), enemas of chloral (20 to 30 grains), and the hot bath.

HYSTERIA.

Definition.—A psychoneurosis characterized by increased impressionability and a lack of self-control, and manifested by a train of symptoms of the most varied character.

Etiology.—Females are especially predisposed, although it occasionally develops in males. It is most common in early adult life. Heredity is an important etiologic factor, the disease frequently being transmitted through hysteric, epileptic, degenerate, or insane parentage. Faulty home-training and education also do much to foster its development.

Traumatism, prolonged emotional excitement, such as worriment, anxiety, disappointment, grief, and all causes that lower the vitality serve to excite it in susceptible individuals.

Pathology.—No causal lesions can be detected after death.

Symptoms.—The various manifestations may be described under three heads: (1) Motor, (2) sensory, and (3) psychic.

Motor Phenomena.—Paralysis not infrequently results from hysteria; it may take the form of a hemiplegia, paraplegia, or monoplegia, although the first is by far the most common. The paralysis is generally paroxysmal, and is frequently associated with contractures and anesthesia. The affected muscles do not waste.

Local paralysis is also common; thus there may be aphonia from paralysis of the vocal cords; dysphagia, from paralysis of the esophagus; and incontinence of urine, from paralysis of the bladder.

Convulsive seizures are common manifestations of hysteria, and may closely simulate the paroxysms of true epilepsy, but there is no aura; the patient usually falls in a comfortable place; consciousness is only apparently lost; the tongue is rarely bitten; the eyes are partially closed; the face is expressive of some emotion; screaming or sobbing is of frequent occurrence; the movements are apt to be tonic, so that the patient assumes the position of opisthotonos, or if clonic, they are apt to be violent and purposive; the seizures are of long duration, and may be continued for several hours, and firm pressure over the ovaries may exaggerate them.

The spasms may be local; thus there may be retention of urine from spasm of the bladder; asthma, from spasm of the bronchi; hiccup, from spasm of the diaphragm; persistent vomiting, from spasm of the stomach; dysphagia, from spasm of the esophagus; and a "phantom tumor," from spasm of the abdominal muscles.

Among other motor phenomena may be mentioned obstinate tremors, choreiform movements, and contractures.

Sensory Phenomena.—There may be a complete loss of sensation in certain parts, as of one side of the body. Anesthesia without other nervous phenomena is usually hysteric. In some cases tactile sensation is preserved and there is a loss only of thermic or painful sensations. The anesthetic part is often unusually pale, and when pricked with a needle, may fail to bleed (ischemia).

The special senses may be involved; thus there may be contraction of the field of vision, complete blindness, loss of smell, loss of taste, or loss of hearing. These palsies are usually transient, and often alterate with one another.

Instead of anesthesia, there may be hyperesthesia or pain. Severe pain in the stomach may simulate gastralgia. An exquisitely painful and tender condition of the abdomen may be mistaken for peritonitis. A localized pain in the head, described as resembling the effect of a nail being driven into it, is termed hysteric *clavus*. The joints occasionally become swollen and tender, resembling arthritis (neuromimesis).

Psychical Phenomena.—Frequently the only conspicuous mental phenomenon is the great lack of will-power, but generally the patients are more or less excitable, highly mercurial, and easily moved to laughter or tears. They frequently manifest a great fondness for sympathy, and this in connection with their weak will-power often leads them to feign symptoms which they really do not have. Among the more serious mental manifestations may be mentioned delirium, ecstasy, catalepsy, and trance.

Treatment.—This must be directed both to the mind and the body, especially to the former. To be successful, the physician must be able to inspire absolute confidence and faith in the mind of the patient. She must be impressed repeatedly with the fact that her condition is a curable one, and that with her thorough coöperation restoration to health will certainly follow. To intimate that her symptoms are feigned or are wholly within her control is a grave error. In many cases no method of treatment proves successful until the patient has been removed from her customary surroundings and separated from her sympathetic relatives and friends.

Suggestion is employed consciously or unconsciously in the treatment of hysteria by every successful physician. Without it many of the remedies recognized as efficacious become wholly impotent. Complete hypnotism, however, is by no means so generally useful as continuous suggestion, and, moreover, in the event of failure, seems capable of still further lowering the will-power and of increasing the emotional excitability.

The physical condition of the patient must not be neglected. General measures, such as hydrotherapy, systematic exercise in the open air, massage, and electricity, are valuable aids to recovery. In grave cases the treatment associated with the name of S. Weir Mitchell often yields admirable results. It consists in isolation from sympathizing friends and relatives; abundant feeding, especially with milk; and complete rest of body and mind, with passive exercise obtained by massage and electricity.

Except to meet underlying conditions and to combat special symptoms, drugs are of little value. Iron and arsenic are useful when there is anemia. Antispasmodics, like valerian, sumbul, asafetida, and camphor, are serviceable in allaying abnormal nervous irritability.

Such combinations as the following may prove useful:

R.	Arseni trioxidi	gr. ss
	Ferri sulphatis exsiccati	
	Extracti sumbul	āā gr. xx
	Asafœtidæ	gr. xl.—M.

Fiant pilulæ No. xx.
SIG.—One three or four times a day.

Or:

℞. Quininæ valeratis
Zinci valeratis
Ferri valeratis . . . . āā gr. xxiv.—M.
Fiant pilulæ No. xxiv.
SIG.—One pill thrice daily.

Occasionally more powerful sedatives, like the bromids, phenacetin, and chloralamid, may be demanded, but the continuous use of such remedies is always to be condemned. Such drugs as morphin, alcohol, and chloral are distinctly dangerous.

When hysteria is complicated by local disease, special treatment will be required, but no operation should ever be performed for the relief of nervous symptoms unless there exists an actual organic lesion.

Convulsions.—Isolation of the patient is imperative. Firm pressure over the ovarian region is often successful. The affusion of cold water over the face is useful. Inhalations of amyl nitrite, or even of chloroform, may be employed if necessary. Strong faradic currents applied to the spine are occasionally efficacious.

Anesthesia is best treated by electricity, especially by the faradic brush. Static electricity, owing to the profound mental impression which it produces, is also useful. *Hyperesthesia* and *pain* often yield to the continuous or interrupted galvanic current. In *paralysis* the patient should be instructed how to regain, by long-continued practice, the use of the affected part. Swedish movements, massage, and faradization are useful adjuvants. In *aphonia* the faradic current, applied by means of special electrodes, is the most reliable remedy. In *contractures* the most useful measures are massage, passive movements, and faradization.

NEURASTHENIA.

(Nervous Prostration.)

Definition.—A functional disease characterized by a lack of nervous energy and increased sensitiveness to external impressions.

Etiology.—The causes are much the same as those that give rise to hysteria. Men are often affected. Overwork, prolonged mental strain, and depressing emotions are common exciting causes.

Symptoms.—*Cerebral Symptoms.*—These include depression of spirits, indisposition, inability to concentrate the mind on one subject for any length of time, insomnia, vertigo, headache, irritability of temper, introspection, and morbid fears.

Spinal Symptoms.—Sometimes these predominate, when the condition is termed *spinal irritation.* The chief manifestations are: Pain in the back, spots of tenderness along the spine, weakness of the extremities, great prostration after moderate exertion, and various subjective phenomena, such as numbness, tingling, formication, and neuralgic pains.

Gastro-intestinal Symptoms.—These consist of anorexia, coated tongue, indigestion, and constipation.

Circulatory Symptoms.—These include palpitation, tachycardia, pseudoangina, cold extremities, and sometimes violent pulsation of the abdominal aorta.

Sexual Symptoms.—In females there is often amenorrhea or dysmenorrhea, and in males impotence or spermatorrhea.

Diagnosis.—The diagnosis is rarely difficult. Before relegating a case to this class, care must be taken to exclude *organic* disease and *such general disorders* as **gout, diabetes,** and **anemia.**

Hysteria.—This affection may be distinguished by the abrupt onset, the intermittent character of the symptoms, and such stigmata as paralysis, anesthesia, contractures, emotional outbreaks, convulsions, and the globus hystericus.

Prognosis.—When the cause can be removed and the patient controlled, the prognosis is favorable.

Treatment.—The treatment is largely hygienic and dietetic, and must vary considerably in different cases. When there has been inactivity, regulated physical exercise will be of great value; on the other hand, the weak and anemic will require rest. In the latter case the plan of treatment introduced by S. Weir Mitchell, and known as the "rest-cure," often gives brilliant results. In all cases careful attention must be given to the diet, bathing, and clothing, and the patient assured that he is suffering from no incurable disease. Frequent bathing with salt water, followed by friction of the skin, will often add to the general vigor. Tobacco and alcohol must be interdicted, and tea and coffee used very sparingly. Tonics, like iron, arsenic, quinin, strychnin, and phosphorus, are often indicated.

ACUTE CHOREA.

(Sydenham's Chorea; Chorea Minor; St. Vitus's Dance.)

Definition.—A nervous affection occurring especially in children, and characterized by irregular, spasmodic movements that increase under excitement and cease during sleep.

Etiology.—The large majority of cases occur in children between the ages of five and fifteen, though adults, especially women during or after pregnancy, are occasionally attacked. More females are affected than males. Heredity sometimes plays an important rôle. Children of a nervous temperament are especially susceptible. Fright or shock is very frequently an exciting factor. In about one-fifth of all cases there is an antecedent history of rheumatism between which disease and chorea there appears to be some relation. Chorea is most common in the spring months.

Pathology.—It is customary to look upon chorea as a neurosis, since no constant lesions have been discovered to account for its clinical manifestations. In some cases emboli in the minute cerebral vessels have been discovered, but their relation to chorea has not yet been determined. A microbic origin has been suggested.

Symptoms.—The first manifestations are usually restlessness and awkwardness in movement. The child cannot remain still, but is constantly raising its shoulders, jerking its head, twisting its fingers, or shuffling its feet. Frequently these symptoms develop so insidiously that the disease is not recognized, and the child is punished for being fidgety.

When the disease is fully established, the disorderly movements become more marked. They may be confined to one member or may involve the entire body. When the facial muscles are affected, the most grotesque expressions are produced; involvement of the arms may interfere with eating and dressing; when the legs suffer, the gait becomes jerking and stumbling; involvement of the larynx causes stammering, and spasm of the muscles of deglutition induces difficult swallowing and choking spells. When the attention is directed to the movements, they invariably grow worse, but they diminish during repose and cease entirely during sleep. Sometimes, in addition to the involuntary movements, there is a distinct loss of power in the affected members. The general health is usually more or less impaired. The child is anemic; the temper is irritable; and the mental power deficient. Auscultation of the heart often detects a murmur that may be an expression either of anemia or of complicating endocarditis.

Chorea Insaniens.—In this form the movements are so violent that the patient is unable to walk, eat, or even to lie in bed. Fever develops, and ultimately the mind becomes delirious. Death frequently results from exhaustion. This form is usually observed in adults, and especially in primiparæ.

Diagnosis.—The recognition of chorea is rarely attended with difficulty. **Disseminated spinal sclerosis** may be distinguished by the presence of nystagmus, a scanning speech, increased reflexes, and a rhythmic tremor that is excited only by movement.

Huntingdon's Chorea.—This disease is usually hereditary; it rarely develops before the age of thirty; it runs a chronic course; and it is characterized by slower and more incoordinate movements than occur in acute chorea, by progressive mental failure, and by a marked suicidal tendency.

Other conditions producing choreiform movements have been considered on page 366.

Complications.—Vegetative endocarditis occurs in from 30 to 40 per cent. of all cases. Pericarditis is occasionally seen.

Prognosis.—In simple chorea recovery usually follows in the course of two or three months. Death from heart complications is a rare termination. Relapses are not infrequent. *Chorea insaniens* frequently terminates fatally through exhaustion.

Treatment.—Rest of body and mind is an essential element of the treatment. The child should be taken from school and placed under the most favorable hygienic conditions. Amusement in the open air when the weather is fine is to be recommended. As the child is generally anemic, iron is indicated in the majority of cases. Among the special remedies arsenic holds the first place. Fowler's solution may be given in doses of two drops thrice daily, gradually increased to eight or ten drops thrice daily. When arsenic fails, cimicifuga should be tried. A dose of 10 minims of the fluid extract may be given after meals and gradually increased to ½ fluidram or more. When the movements are very violent and interfere with sleep, recourse must be had to chloral, bromids, hyoscin, or morphin, but these drugs should not be used unless the symptoms are of great severity.

Chorea Insaniens.—Powerful sedatives like hyoscin, chloral, and morphin are required to allay the violent excitability and jactitation. Inhalations of chloroform are sometimes useful. Stimulants are almost always required. When the patient is unable to swallow, no time should be lost in resorting to forced feeding. Severe cases of chorea complicating pregnancy will call for the induction of premature labor.

NEURALGIA.

Definition.—A functional disease of the nerve-trunks, characterized by paroxysms of intense pain.

Etiology.—It is a disease of adults. Women are much more frequently affected than men. Heredity is an important etiologic factor. It is frequently an expression of anemia. It may result from the action of some toxic agent in the blood; thus it is common in malaria, rheumatism, gout, syphilis, and chronic lead-poisoning. It may be caused by reflex irritation; thus a trifacial neuralgia may depend on caries of the teeth or eye-strain. In some cases neuralgia results from organic disease of the nerve-center; thus obstinate trifacial neuralgia may be dependent upon some degeneration or tumor of the Gasserian ganglion.

Exposure to cold and wet frequently acts as an exciting cause in susceptible persons.

Pathology.—The pathologic condition upon which neuralgia depends is unknown. In many cases, no doubt, it is a manifestation of neuritis.

Symptoms.—Certain prodromes frequently give warning of an approaching attack; these are chilliness, depression of spirits, and perhaps tingling in the part to be affected. The chief symptom is intense pain, which is usually of a sharp, stabbing character. The area supplied by the affected nerve is generally hyperesthetic, and palpation may detect spots of exquisite tenderness where the nerve makes its exit through a bony canal or fibrous sheath —*points douloureux* of Valleix. In some cases the pain is attended with tremors or spasms of the muscles. Inspection of the part usually reveals nothing abnormal, but occasionally distinct swelling is observed.

The attack lasts from a few minutes to many hours, and its subsidence may be marked by the passage of a large amount of pale urine. The interval between the paroxysms varies in different cases; it is frequently several weeks or months. It is noteworthy that the attacks often recur at regular intervals.

Trifacial Neuralgia (*Tic Douloureux; Prosopalgia*).—In this variety the pain involves one or more branches of the trifacial nerve. The tender points correspond to the supra-orbital, infra-orbital, and mental foramina. Violent spasms of the muscles are frequently observed. In long-standing cases the hair on the affected side may become coarse and bleached.

Intercostal Neuralgia.—In this variety the pain follows the course of the intercostal nerves. It is frequently associated with an eruption of herpes zoster. Spots of tenderness may be detected near the vertebral columns, in the middle of the nerve, and near the sternum. The possible dependence of intercostal neuralgia upon spinal caries or thoracic aneurysm must not be forgotten.

Occipital neuralgia involves the upper cervical nerves. A spot of tenderness may be discovered midway between the mastoid process and the upper cervical vertebræ. This form of neuralgia may also be an expression of spinal caries.

Sciatica has been described elsewhere.

Diagnosis.—**Neuritis.**—The continuous pain, the tenderness along the entire nerve, the presence of paresthesia, anesthesia, paresis, and wasting will serve to distinguish neuritis from neuralgia.

The **lightning-pains of locomotor ataxia** must not be mistaken for neuralgia. The abolished patellar reflex, the loss of coördination, and the Argyll-Robertson pupil in the former will indicate the diagnosis.

Prognosis.—For the attack the prognosis is good; for permanent cure, it must be guarded. When the cause can be removed, the prognosis is favorable.

Treatment.—*The Interval.*—Careful search should be made for an exciting cause, which, if found, must be removed. The teeth, eyes, nose, gastro-intestinal tract, urine, and blood should be carefully examined.

When the disease is associated with anemia, iron and arsenic will be indicated. If there is any suspicion of syphilis, mercury and iodids should be tried. When a malarial element is present, quinin may effect a cure. When rheumatism is an etiologic factor, salicylates and alkalis may prove beneficial. In gouty subjects much may be expected from regulation of diet, systematic exercise, and the administration of alkalis. In chronic lead-poisoning iodids are indispensable.

All influences that tend to induce a morbid excitability of the nerves or of their centers—mental or physical fatigue, emotional excitement, sexual excess, overindulgence in tobacco, tea, coffee, or alcohol—should be removed as far as possible.

In every case we must endeavor to improve the general nutrition, which is almost always disturbed. The measures to be employed for this purpose include an abundance of fresh air, proper food, regular hours, adequate protection from the vicissitudes of weather, systematic exercise, frequent bathing with friction, and the use of such tonics as iron, arsenic, cod-liver oil, and hypophosphites.

The following combination is often useful:

℞.	Quininæ sulphatis	gr. xxiv
	Arseni trioxidi	gr. ss
	Ferri reducti	ʒss
	Calcii glycerophosphatis	ʒiss.—M.

Pone in capsulas No. xxiv.
Sig.—One capsule after meals.

Finally, when all other measures fail, recourse may be had to surgical interference. Nerve-stretching and nerve-section are the operations usually performed. Lasting benefit, however, is rarely obtained from either operation. In obstinate trifacial neuralgia removal of the Gasserian ganglion usually affords permanent relief, though the operation is not without danger.

The Attack.—Heat, dry or moist, may be applied for its soothing effect. A liniment of aconite or of chloroform is sometimes efficacious. Menthol and chloral camphor are useful in neuralgia of superficial nerves when the pain is slight. Acupuncture and aquapuncture are effective, but are not suitable for use about the face. In obstinate cases active counterirritation by means of blisters or the thermocautery will be found a potent remedy. In trifacial neuralgia the blisters may be applied behind the ear.

Among the internal remedies most worthy of confidence may be mentioned phenacetin, antipyrin, acetanilid (5 to 10 grains), bromids, cannabis indica, croton chloral, caffein, gelsemium, and salicylic compounds. Morphin is undoubtedly the most certain means we possess of affording temporary relief, but on account of the danger of inducing the opium habit it should be employed only as a last resort. Combinations of a bromid with phenacetin may often be prescribed advantageously. Croton chloral, in doses of from 5 to 10 grains, and tincture of gelsemium, in doses of 10 minims or more, are occasionally serviceable in trifacial neuralgia. Caffein (3 to 5 grains) is often efficacious. Combinations of caffein with phenacetin or with bromids in many cases do more good than single drugs. Neuralgia brought on by exposure to cold and wet is favorably influenced by the salicylates. In such, the following combination will be found of value:

℞. Acetphenetidini
Salophen . . . . āā ʒiss
Codeinæ sulphatis . . . . gr. iij.—M.
Fiant chartulæ No. xij.
SIG.—One powder every two or three hours.

MIGRAINE.

(Hemicrania; Megrim; Sick-headache.)

Definition.—A neurosis characterized by periodic attacks of intense headache, usually unilateral and often associated with visual, gastric, and vasomotor disturbances.

Etiology.—It is frequently hereditary. It is more common in women than in men. It usually develops in early life. Anemia, gastric disturbances, gout, eye-strain, menstrual disorders, overwork, and prolonged excitement predispose to it.

Symptoms.—Prodromes, such as restlessness, depression, and malaise are common. The attack is often ushered in with visual disturbances, such as flashes of light, dimness of vision, or hemianopsia. The pain is severe and generally limited to the temporofrontal region of one side, though it sometimes spreads until it involves the whole head. The patient is very sensitive to light and sound, and during the attack usually confines herself to a darkened room. Nausea and vomiting are frequently present. In some cases the temporal artery is contracted, the face is pale, and the pupil large; in others the artery is dilated, the face is flushed, and the pupil small. The duration of the attacks varies from a few hours to several days. In the intervals, which are often of definite duration, the patient may be quite well.

Less frequent symptoms are vertigo, hallucinations of sight, cramps of the facial muscles, tingling or numbness in one hand, partial aphasia, and paresis of the ocular muscles.

Prognosis.—Perfect cure is rare, but many cases are improved by treatment. The attacks often cease spontaneously at middle life.

Treatment.—In the interval the treatment is that of neuralgia. Cannabis indica is sometimes of value. From a quarter to a half a grain of the extract may be given for several weeks. Little recommends:

℞. Sodii arsenatis	gr. ij
Extracti cannabis indicæ	gr. vj
Extracti belladonnæ	gr. viij.—M.

Fiant pilulæ No. xxiv.
SIG.—One pill twice daily.

The Attack.—The patient should be kept at rest in a quiet, darkened, well-ventilated room. Antipyrin, phenacetin, caffein, bromids, and salicylic compounds are the most useful remedies. They may often be combined with advantage, as in the following formulas:

℞. Caffeinæ citratæ	gr. xij
Acetphenetidini	ʒj
Sodii bromidi	ʒij.—M.

Fiant chartulæ No. xij.
SIG.—One powder every two hours.

Or:

℞. Salophen	ʒiss
Acetphenetidini	ʒj
Caffeinæ citratæ	gr. xij.—M.

Fiant chartulæ No. xij.
SIG.—One every two hours.

PARALYSIS AGITANS.

(Parkinson's Disease; Shaking Palsy.)

Definition.—A chronic nervous disease, characterized by a gradually spreading tremor, muscular weakness and rigidity, and a peculiar gait, termed festination.

Etiology.—Advanced life, a neuropathic tendency, mental strain, heredity, and exposure to cold and wet are predisposing factors. It sometimes develops suddenly after intense mental or emotional excitement.

Pathology.—The pathology is unknown. The lesions found—degeneration of arterioles, perivascular sclerosis, pigmentation of ganglionic cells—are similar to those induced by senility.

Symptoms.—In some cases the onset is abrupt, but more commonly the disease develops insidiously. A tremor appears, usually in the fingers, and gradually spreads until it involves all the extremities and sometimes the neck and head. At first the tremor may be paroxysmal, but as the disease advances it becomes almost continuous. Excitement increases it, but it is notworthy that physical effort temporarily diminishes or checks it. The face becomes expressionless and the speech slow and measured. Later, muscular rigidity develops, and the head is bowed, the body bent forward, the arm flexed, the thumbs turned into the palms and grasped by the fingers, and the knees slightly bent. At this time the gait is characteristic; the steps grow faster and faster, the body inclines more and more forward until the patient falls, finds support in some neighboring object, or straightens himself by a supreme effort of the will. The term *festination* has been applied to this peculiar gait. Occasionally a tendency to fall backward—*retropulsion*—replaces festination. The rigidity and muscular weakness render all movements slow and stiff.

Intelligence is usually good. There is no anesthesia, but there are various manifestations of paresthesia, such as numbness and tingling and a sensation of heat. In some cases free perspiration has been observed.

Diagnosis.—The tremor, rigidity, weakness, flexion of the body and members, lack of facial expression, and festination are the diagnostic features. In some cases the tremor is absent. Paralysis agitans must be distinguished from **disseminated sclerosis.** In the latter the tremor is absent when the patient is quiet, and is made worse by efforts to control it; cerebral symptoms are generally present; nystagmus is often noted; and the attitude and gait are entirely different from those of paralysis agitans.

Prognosis.—Recovery rarely, if ever, occurs. In some cases, after reaching a certain point, the disease remains stationary. The progress is slow and the duration indefinite.

Treatment.—Measures intended to improve the tone of the system are indicated; these are: A regulated diet, rest of body and mind, frequent bathing followed by friction of the skin, and the use of some tonics as iron, arsenic, and phosphorus. The rigidity and tremors are sometimes improved by massage and electricity. Among the remedies recommended for the tremors are bromid of potassium, hyoscyamin ($\frac{1}{100}$ grain), hyoscin ($\frac{1}{125}$ grain), and duboisin ($\frac{1}{100}$ to $\frac{1}{60}$ grain), but the improvement following their use is only slight and temporary.

ARTISANS' CRAMP.

Definition.—A spasmodic affection of the muscles induced by prolonged work requiring delicate coördination, and occurring only in the performance of that particular work.

Etiology.—It is more common in men than in women, and the nervous temperament predisposes to its development. The occupations in which it is most apt to occur are writing, piano-playing, sewing, and telegraphing.

Pathology.—The disease is evidently not peripheral, for when the other hand is substituted, the condition soon develops in that member. It is probably dependent upon unnatural irritability of the nerve-centers.

WRITERS' CRAMP.

(Graphospasm; Scriveners' Palsy.)

Symptoms.—The condition usually begins with a sense of fatigue, weight, or actual pain in the affected muscles. Soon the fingers are seized with a tonic or clonic spasm whenever the pen is grasped (spastic form). In some cases the hand when put into use becomes the seat of a decided tremor (tremulous form); in a third group of cases the chief phenomena are excessive weakness and fatigue which disappear as soon as the pen is laid aside (paralytic form).

Prognosis.—Guardedly favorable. The disease is obstinate, but cure generally follows protracted rest.

Treatment.—Absolute rest is the essential element of treatment. The general condition should be improved by iron, arsenic, strychnin, and cod-liver oil. Massage, electricity, and passive movements give good results.

TETANY.

(Tetanilla; Intermittent Tetanus.)

Definition.—A comparatively rare disease characterized by continuous or intermittent tonic spasms, especially of the extremities.

Etiology.—Tetany is a disease chiefly of infants and young adults. In infants it is usually associated with rickets, gastro-intestinal disorders, or the specific fevers. In adults it has developed in gastro-intestinal infections, especially gastrectasis, in acute infectious diseases, in pregnancy or lactation, in poisoning by certain drugs, such as chloroform or morphin; or it has followed thyroidectomy. An epidemic form has also been described, but some of the outbreaks seem to have been hysteric.

Pathogenesis.—There is reason to believe that the disease is due to certain endogenous poisons which normally are neutralized by the secretion of parathyroid glands.

Symptoms.—The patient is seized with bilateral tonic spasms, beginning in the hands or feet and spreading upward. In many cases all four extremities are attacked. The muscles of the trunk and face are sometimes involved, but those of the jaw only rarely. The spasms are usually intermittent, though they may be continuous and last for days or even weeks. They are sometimes attended by pain. Laryngismus stridulus is not uncommon in children. As was pointed out by Trousseau, pressure on the nerve-trunks and blood-vessels of the affected limb will reproduce the contractions. Sometimes the mechanical irritability of the motor nerves is so increased that a mere tap will excite spasm (Chvostek's phenomenon). The electric irritability of the motor nerves and muscles is also increased (Erb's phenomenon). Not rarely the cramps are accompanied by slight fever and edema of the hands and feet.

Diagnosis.—In **tetanus** a source of infection is usually found; trismus or lockjaw appears early; and the muscles of the back are more contracted than those of the limbs.

Hysteria may be distinguished from tetany by the history, the emotional disturbances, the unilateral character of the spasms, and the absence of Trousseau's sign.

Prognosis.—The outlook is generally good, though in tetany associated with gastrectasis or following thyroidectomy the mortality is high.

Treatment.—The cause should be sought for and removed if possible. Thyroid and parathyroid extracts have been very serviceable in some cases. Surgical intervention is indicated when there is gastrectasis. Warm baths are useful. Among the sedatives, the bromids are most serviceable. In severe cases it may be necessary to use hyoscin, chloral, or morphin.

THOMSEN'S DISEASE.

(Congenital Myotonia.)

Definition.—A disease confined to certain families, and characterized by tonic spasms of the muscles, induced by voluntary movements.

Etiology.—The disease is usually congenital, and transmitted from one generation to another. Several members of the same family are commonly affected.

Pathology.—Unknown.

Symptoms.—The disease appears in early childhood, and is manifested by tonic spasm of the muscles upon every attempt at voluntary motion. This is especially marked after periods of inactivity. In a few moments the rigidity wears away and the movements become free. From repeated contractions the muscles become firm and extremely well developed. Under electric stimulation the muscles contract and relax slowly. The disease is incurable, but shows no tendency to prove fatal.

RAYNAUD'S DISEASE.

(Symmetric Gangrene.)

Definition.—A vasomotor neurosis characterized by local anemia, congestion, and gangrene.

Etiology.—The cause is unknown. The disease is believed to be dependent upon spasm of the peripheral arterioles.

Symptoms.—In the first stage the affected part becomes extremely pale, cold, and anesthetic (*local syncope*). After a variable time the part becomes purple, livid, and intensely painful (*local asphyxia*). Such attacks may be excited by cold, and come and go without damaging the part. Occasionally the disease advances to the third stage, in which congestion gives way to *dry gangrene*. Symmetric parts, as a finger on each hand, a toe on each foot, or the lobes of the ears, are usually affected. Hemoglobinuria may occur in, or replace, an attack.

Prognosis.—The attacks persist, but life is rarely endangered.

Treatment.—Patients liable to attacks should be well protected against cold. Tonics are often indicated. Frequent bathing followed by friction is useful. Raynaud advises the use of the continuous current—one pole over the spine and the other over the affected area. Nitroglycerin may prove useful.

ACUTE ANGIONEUROTIC EDEMA.

Definition.—A neurosis characterized by transient circumscribed edema developing without obvious cause.

Etiology.—Beyond a distinct hereditary tendency nothing is known of its cause. According to Quincke, there is a temporary vasomotor dilatation of the vessels, followed by the transudation of serum.

Symptoms.—Edematous swelling suddenly appears in some part of the body, particularly in the face and hands. Coincident with the edema there may be marked gastrointestinal symptoms, such as vomiting, gastralgia, and colic. The disease is allied to urticaria and the latter may precede the outbreak.

The attacks usually occur at intervals of a few weeks.

Prognosis.—The disease generally proves very obstinate, but unless it involves the larynx, it does not endanger life.

Treatment.—General tonics, like iron, quinin, and strychnin, are sometimes useful.

TROPHIC DISORDERS, SUNSTROKE, AND INTOXICATIONS.

MUSCULAR DYSTROPHIES.

Definition.—An atrophic condition of the muscles developing in early life and not dependent upon any lesion in the nervous system.

Etiology.—The disease usually manifests itself before puberty. It is more common in males than in females. It is frequently transmitted from generation to generation, and several members of the same family may be similarly affected.

Pathology.—No lesion in the cord or nerves is observed. Gowers regards the disease as of developmental origin. Microscopic examination of the muscles reveals atrophy of their fibers and an unnatural amount of fat and connective tissue. When the latter elements are considerably increased, a pseudohypertrophy results (pseudomuscular hypertrophy).

Varieties.—The following types are recognized: (1) Pseudomuscular hypertrophy; (2) Erb's juvenile dystrophy; (3) Landouzy-Dejerine type.

Pseudomuscular Hypertrophy.—This form begins in children between the second and seventh year. The first symptom to attract attention is weakness of the muscles; the child is awkward, stumbles, and in walking seeks support. As the paralysis increases the muscles, particularly those of the calf, thigh, buttock, and back, enlarge. The upper extremities are less frequently affected. When the child assumes the erect posture, the feet are wide apart, the belly protrudes, and the spinal column shows a marked curvature with the convexity forward. The manner of rising from the recumbent position is characteristic: He straightens himself either by grasping the knees or by resting the hands on the floor in front of him, extending the legs, and pushing the body backward. The gait is waddling in character.

The electric contractility of the muscles is gradually reduced, but the reaction of degeneration is not present. There are no fibrillary twitchings in the muscles. The knee-jerk is lessened or abolished. There are no mental or sensory disturbances.

In the course of a few years the paralysis becomes so marked that the patient is unable to leave his bed; the enlargement of the muscles is followed by atrophy; and finally death results from some intercurrent disease or inflammation of the lungs induced by the weakened respiratory power.

Erb's Juvenile Dystrophy.—This form usually develops between the ages of twelve and sixteen. The muscles of the shoulder are first affected. The wasting may be preceded by hypertrophy.

In *chronic poliomyelitis* (progressive muscular atrophy of spinal origin), the symptoms come on later in life, hereditary or family influences are rarely present, the small muscles of the hand are first affected, and the wasting is associated with fibrillary twitchings. In *multiple neuritis* paralysis precedes the wasting, sensory symptoms are usually present, and the history reveals a definite cause.

Landouzy-Dejerine Type.—This type usually develops in early childhood and is characterized by bilateral atrophy of the facial muscles. It differs from *bulbar palsy* in that it does not involve the tongue or the muscles of deglutition.

Prognosis and Treatment.—The disease is curable, but the progress is slow. Massage, electricity, and graduated exercise may be followed by temporary improvement.

FACIAL HEMIATROPHY.

(Unilateral Progressive Atrophy of the Face.)

Definition.—A rare affection, characterized by progressive wasting of tissues—bones and soft parts—on one side of the face.

Etiology.—The disease usually develops in childhood. It has been excited by injury of the face.

Pathology.—In the few cases examined chronic trigeminal neuritis has been discovered.

Symptoms.—The first phenomenon is often discoloration of the skin; this is soon followed by a slow wasting of all the tissues on the affected side of the face. The hair falls, the eye recedes, and the teeth drop out.

Prognosis.—The disease is progressive and incurable.

ACROMEGALY.

(**Marie's Disease; Pachyacria.**)

Definition.—A nutritional disease, characterized by enlargement of the bones and overlying tissues, chiefly of the hands, feet, and face.

Etiology.—Unknown. It usually develops in early life. Marie attributed it to a loss of function of the pituitary body.

Pathology.—Examination of the bones reveals a true hypertrophy, particularly of the cancellous structures. In many cases the pituitary body has been found to be the seat of simple hypertrophy, degeneration, adenoma, or sarcoma; in a few the thymus or thyroid gland has been diseased.

Symptoms.—The hands and feet are considerably enlarged, especially in breadth; the fingers and toes are stumpy and the nails are flat and small. Hypertrophy of the inferior maxillary bone leads to elongation of the face and protrusion of the lower jaw. The lips are large and everted. Among occasional symptoms may be mentioned spinal curvature, polyuria, glycosuria, persistent headache, deafness, blindness from atrophy of the optic nerve, loss of sexual power, and, in women, menstrual disorders.

Diagnosis.—Acromegaly might be mistaken for **myxedema**, but in the latter only the soft parts are involved; the skin is firm and adherent, instead of soft and mobile, as in acromegaly; and the face is round.

In **Paget's osteitis deformans** the long bones are especially involved, and are not only enlarged, but considerably deformed, and the face has a peculiar triangular shape.

Prognosis.—The affection is incurable, but the duration may be indefinite. Acute cases lasting two or three years are usually associated with sarcoma of the pituitary body.

Treatment.—So far, remedies have been futile.

SUNSTROKE.

(**Heat-stroke; Thermic Fever; Coup de Soleil; Insolation; Heat-exhaustion.**)

Definition.—An affection resulting from exposure to excessive heat.

Varieties.—Two varieties are observed: thermic fever and heat-exhaustion.

THERMIC FEVER.

Etiology.—The exciting cause is exposure to intense heat, natural or artificial. Bodily fatigue and intemperance are important predisposing factors. It is probable that the excessive heat leads to the production of toxic substances that disturb the heat-regulating centers in the brain.

Pathology.—After death from thermic fever rigor mortis develops early and is marked. The various organs, especially the brain, are deeply congested. The left ventricle is firmly contracted, and the right is dilated and filled with blood. The blood is dark and uncoagulated. Microscopic examination of the tissues reveals parenchymatous degeneration or cloudy swelling.

Symptoms.—Prodromes are frequently present and consist of exhaustion, vertigo, nausea, and headache. These symptoms are followed by coma, and in this state the face is flushed; the eyes are injected; the skin is dry and burning; the temperature ranges from 106° to 112° F.; the pupils are contracted; the respirations are rapid and noisy; and the pulse is full and rapid. Unless the temperature soon falls, the respirations become shallow, the pulse weakens, and death results in a few hours. There is a very malignant form in which the patient is suddenly stricken comatose and dies in a few hours from cardiac failure.

Sequelæ.—They include chronic meningitis; epilepsy; insanity; failure of memory; and extreme sensitiveness to high temperature.

Diagnosis.—The conditions under which the coma has developed, together with the extremely high temperature of the body, will serve to distinguish sunstroke from apoplexy, alcoholism, and uremia.

Prognosis.—This should be very guarded. Probably 40 per cent. perish.

Treatment.—The patient should be promptly placed in a bath of iced water and should be rubbed with ice. Ice-water enemas are also useful. The subcutaneous or intravenous injection of normal salt solution has proved efficacious in some cases. Packard and others have found blood-letting (10 to 20 ounces) very effective in some cases. Feebleness of the pulse is not necessarily a contra-indication, as the circulation often improves during the operation.

HEAT-EXHAUSTION.

Pathology.—According to Wood, heat-exhaustion depends on a vasomotor paresis, as a result of which there is a determination of blood from the brain and surface of the body to the great blood-vessels of the abdomen.

Symptoms.—The mind is dazed, but consciousness is not lost; the surface is pale and cold; the skin is moist; the respirations are shallow and hurried; and the pulse is rapid and feeble.

Prognosis.—Recovery generally follows under appropriate treatment.

Treatment.—The patient should be covered with hot blankets, and hot bottles should be placed near the feet. Brandy, ammonia, and strong coffee are useful stimulants. Strychnin hypodermically is an efficient remedy.

ALCOHOLISM.

(Inebriety.)

Acute Alcoholism.—The ingestion of large quantities of alcohol produces the following symptoms: Flushing of the face, mental excitement, quickening of the pulse and respiration; then incoherent speech, delirium, dilated pupils, loss of coördination, subnormal temperature, vomiting, and, finally, stupor and coma. Not infrequently the coma is interrupted by convulsive seizures. In most cases, if the dose has not been too large, recovery follows in a day or two.

Chronic alcoholism is characterized by disturbed sleep, fine tremors, mental impairment, injection of the eyes, redness of the nose, and the symptoms of gastro-intestinal catarrh. When the habit is long continued, degenerative and cirrhotic changes in the heart, blood-vessels, liver, and kidneys are apt to develop.

A common complication of chronic alcoholism is *delirium tremens*, which is generally excited by temporary excess, an injury, or some acute intercurrent disease, especially pneumonia. It is manifested by great mental excitement, insomnia, incoherent speech, tremors, disordered intellect, and terrifying hallucinations of sight or hearing. The pulse is rapid and feeble, the appetite is lost, the bowels are constipated, and the temperature is slightly elevated. In favorable cases convalescence follows in a few days, but not infrequently typhoid symptoms develop and the attack ends in death.

Among other sequels of dipsomania may be mentioned pneumonia, chronic meningitis, multiple neuritis, amblyopia, epilepsy, and dementia.

Diagnosis.—The *coma of alcoholism* must be distinguished from the coma of other diseases. The history, the absence of paralysis, the subnormal temperature, the fact that the patient can be aroused by screaming in the ear or by firm pressure over some sensitive spot like the supra-orbital notch, the odor on the breath, and the absence of other cause will usually prevent an error in diagnosis.

Delirium tremens is recognized by the history, restlessness, delirium, tremors, and terrifying hallucinations.

The tremors of chronic alcoholism may be recognized by the history, the associated evidence of alcoholism, and by the fact that they are worse in the morning and improve after the use of the stimulant.

Prognosis.—*In acute alcoholism* the prognosis should be guardedly favorable. *In delirium tremens* recovery generally follows unless there is great debility. *In alcoholic pneumonia* the outlook is grave; recovery is exceptional.

In alcoholic neuritis the symptoms usually subside under appropriate remedies and abstinence from the stimulant.

In *chronic alcoholism* the prognosis is generally unfavorable. When the habit is fully established, it is rarely permanently broken; temporary improvement is only too often followed by a relapse.

Treatment.—*Acute Alcoholism.*—The stomach should be emptied by the stomach-pump, a stimulating emetic, or the hypodermic injection of apomorphin ($\frac{1}{10}$ grain). If the coma persists and the pulse weakens, cardiac stimulants, like ammonia, strychnin, and digitalis, should be administered hypodermically. Douching and flagellation may also be employed to arouse the patient.

Treatment of Delirium Tremens.—As there has usually been a complete abstinence from food during the debauch leading to the delirium, nutritious foods are always necessary, and the best are milk with lime-water and highly seasoned beef-tea. Sleep must be secured by chloral (20 grains), hyoscin ($\frac{1}{100}$ grain), potassium bromid (1 dram), or paraldehyd ($\frac{1}{2}$ to 1 fluidram). Active catharsis should be encouraged. When the pulse is weak, strychnin and digitalis will be found useful stimulants. In many cases physical restraint will be required; it may be secured by strapping the patient to the bed with sheets. Should profound stupor develop, the application of a blister to the back of the neck or a few light touches of the actual cautery will often serve to arouse the patient.

Chronic Alcoholism.—It is necessary that alcohol shall be withdrawn; the rapidity with which this can be accomplished will depend on the circumstances. In most cases the temptation to drink is so strong that confinement in an inebriate asylum is essential to the success of the treatment. Various substitutes have been recommended for alcohol, among which may be mentioned bromid of potassium, chloral, cocain, hyoscin, and cannabis indica. As a rule, they accomplish little beyond quieting the patient and occasionally securing sleep. The diet should be nutritious, and carefully adapted to the condition of the stomach, which is usually the seat of chronic catarrh. Tonics, like iron, quinin, and strychnin, are often indicated. Graduated physical exercise is sometimes of decided value.

CHRONIC OPIUM POISONING.

(**Morphinomania.**)

Symptoms.—The symptoms resulting from the habitual use of opium are an irresistible craving for the drug, loss of flesh and strength, tremors, anemia, a peculiar sallow complexion, anorexia, deranged digestion, a tendency to diarrhea, disturbed sleep, mental depression, irritability, and a characteristic propensity for lying and deceiving.

Treatment.—Isolation in a special institution or asylum is almost imperative. As a rule, the drug should be withdrawn rapidly, but in aggravated cases not too abruptly, for fear of collapse. The diet should consist of nutritious, easily digested food. Strychnin, while it is without specific action, is often extremely valuable for its stimulating effect. Bromids and cannabis indica are sometimes useful in ameliorating the distress that follows the withdrawal of opium. Sulphonal, paraldehyd, and chloretone are the best hypnotics. Massage, graduated exercise, and the Turkish bath are useful roborant measures in the convalescent stage.

CHRONIC LEAD-POISONING.

(**Plumbism; Saturnism.**)

Etiology.—Chronic lead-poisoning may be brought about by the too prolonged use of the salts of lead for medicinal purposes, but it is much more frequently induced in workmen who are exposed to the fumes or dust of lead, or who handle the metal or paints containing it. It may follow, also, the accidental introduction of lead into the system through drinking-water, articles of food, hair-dyes, and cosmetics. Occasionally it results from the use of water that has been carried through lead pipes or has been stored in cisterns lined with lead.

Pathology.—The muscles are degenerated, and the peripheral nerves frequently reveal evidences of chronic neuritis. In cases associated with marked muscular atrophy there may be also degeneration of the ganglionic cells in the gray horns of the spinal cord.

Symptoms.—The following are the chief manifestations: Anemia with granular degeneration (basophilic) of the red cells; severe colicky pains centering around the umbilicus, with retraction and rigidity of the abdominal walls; constipation; a blue line on the gums near the insertion of the teeth, due to the deposition of a sulphuret of lead; paralysis; tremors; intense headache; pains in the joints (arthralgia); arteriosclerosis; chronic interstitial nephritis; and grave cerebral symptoms (encephalopathies).

The Paralysis.—This usually attacks the muscles supplied by the musculospiral nerve,—the extensors of the fingers and of the wrist,—causing the so-called "wrist-drop." The affected muscles ultimately atrophy and yield the reactions of degeneration. Occasionally other muscles are involved, such as the extensors of the legs, the recti of the eye, and the adductors of the larynx. Sensation is not affected.

Encephalopathies.—These are among the more rare manifestations of plumbism, and consist of convulsions, coma, delirium, intense headache, and blindness from atrophy of the optic nerves.

Prognosis.—Guardedly favorable.

Treatment.—Prophylaxis consists in absolute cleanliness; the use of respirators in lead factories; the avoidance of eating in an atmosphere laden with the dust of the metal; and in the occasional use of Epsom salts.

The curative treatment consists in the administration of iodid of potassium (5 to 10 grains thrice daily) and the use of sulphur baths. Constipation should be relieved by Epsom salts. The colic may require the hypodermic injection of morphin and atropin and the application of hot fomentations to the abdomen. The paralysis generally yields to massage, the constant current, and hypodermic injections of strychnin.

DISEASES OF THE SKIN AND ITS APPENDAGES.

THE COLOR OF THE SKIN.

Pallor as a *permanent condition* is generally an expression of anemia; but it should be borne in mind that in some cases the surface is pale when the blood is normally rich in corpuscles and hemoglobin; and that in other cases the surface has a natural color when the blood is considerably deficient in corpuscles and hemoglobin. It follows, therefore, that an absolute diagnosis of anemia must rest on an analysis of the blood.

Pallor as a *temporary condition* may result from emotional excitement, exposure to extreme cold, shock, syncope, or collapse.

Yellowness of the skin may result from *jaundice*, in which case the conjunctivæ will also be yellow and the urine will contain bile. Yellowness may also result from *chlorosis or pernicious anemia*, and in these cases the normal color of the conjunctivæ, the associated symptoms of the disease, and the absence of bile in the urine will indicate the cause.

Whiteness of the Skin.—A milk-white hue over extensive areas may be observed in *albinism*, *vitiligo*, and in *leprosy*.

Dark-brown or gray discoloration of the skin is observed in the following conditions:

Addison's Disease.—In this affection the skin has a bronzed appearance, which is especially marked on exposed parts; the buccal mucous membrane may also reveal discolored plaques, and there are, in addition, anemia, prostration, and gastric irritability.

Argyria.—This term is applied to the dark-gray discoloration of the exposed parts that follows the prolonged use of nitrate of silver. The discoloration is due to a deposition of the oxid of silver and is more or less permanent. It is said to be preceded by a dark line on the gums, similar to the one observed in chronic lead-poisoning. Formerly, when nitrate of silver was used extensively in the treatment of epilepsy, it was not an uncommon condition.

Vagabondismus.—This term is applied to the dark-brown discoloration of the skin that follows prolonged exposure to the weather, uncleanliness, and perhaps the irritation of the skin resulting from pediculosis.

Blueness of the skin as a permanent condition is generally an expression of cyanosis.

HARDNESS OR INDURATION OF THE SKIN.

Induration of the skin is observed in *scleroderma*. In this affection the skin is tense, hide-bound, and more or less pigmented. Induration is also observed in *myxedema*. In this condition the skin is swollen, as in edema, but it is firm, inelastic, and does not pit on pressure. In addition the features are peculiarly broadened and the mental power is impaired. Circumscribed patches of induration are observed in *morphea*. The circumscribed patches, with hyperemic or pigmented borders, and the smooth, shiny, atrophied condition of the skin are the diagnostic features.

Edema, or *dropsy of the subcutaneous tissues*, when extreme, may also cause induration.

A brawny, indurated condition of the muscles, especially of the legs, is frequently observed in *scurvy*. It probably results from a sanguineous exudation. The anemia, purpuric spots, and spongy, bleeding gums will aid in the diagnosis.

GLOSSY SKIN.

"*Glossy Skin.*"—This term was applied by Paget to indicate a smooth, atrophied, and shiny appearance of the skin. It is most frequently observed after inflammation or injury of the nerve-trunks. It is sometimes associated with an intense burning pain to which Mitchell has given the name *causalgia.*

ENLARGEMENT OF THE SUPERFICIAL VEINS.

Enlargement of the superficial veins may result from chronic heart, lung, or liver disease or from the pressure of a tumor or aneurysm on deep-seated veins. As a general condition it may be congenital and result from occlusion of the deep veins.

"Caput Medusæ."—This term is applied to a circle of dilated veins surrounding the umbilicus. It is indicative of obstruction to the portal circulation, and may result from atrophic cirrhosis of the liver, from thrombosis of the portal vein, or from the pressure of a tumor on the portal vein.

CUTANEOUS EMPHYSEMA.

Cutaneous emphysema consists in an escape of air into the cellular tissue. It is manifested by a diffuse, pallid swelling of the skin, which crackles on palpation and which pits on pressure; but unlike edema, the depression immediately disappears when the finger is withdrawn. It may result—(1) From traumatism of the lungs, as in gunshot wounds of the chest or fracture of the ribs. (2) From rupture of the esophagus, stomach, intestines, larynx, trachea, or lungs. The rupture of these organs is usually due to ulceration, such as may occur in cancer of the esophagus, tuberculous cavity of the lung, or purulent pleurisy.

ABNORMAL CONDITIONS OF THE NAILS.

Atrophy of the Nails.—The nails may become dry, brittle, discolored, and cracked in organic disease of the spinal cord; after inflammation or injury of the peripheral nerves; after prolonged febrile diseases, like typhoid fever; and in certain affections of the skin that involve the matrix of the nail, as eczema, psoriasis, and ringworm.

Curving of the Nails.—Incurvation of the nails is generally associated with clubbing of the terminal phalanges. It is observed in phthisis, chronic cardiac disease, and in many wasting diseases.

Onychia.—Inflammation of the matrix of the nail may result from injury; from syphilis; from organic disease of the spinal cord, as locomotor ataxia; from arthritis deformans; and from cutaneous affections involving the matrix, as leprosy, ringworm, and eczema.

CUTANEOUS ERUPTIONS.

Macules.—Macules are discolored spots that are neither elevated nor depressed.

A general red macular eruption is observed in the following conditions:

Syphilis.—Secondary syphilis may manifest itself as an eruption of small red macules. They are usually abundant and frequently cover the entire body; they lack subjective symptoms; they are usually associated with the history or with the evidences of syphilis, such as the scar of the chancre, bone-pains, alopecia, swollen glands, and sore throat.

Erythema multiforme may manifest itself as a macular eruption, but the macules are usually associated with dark-red papules or tubercles. The multiformity of the lesions; their preference for the extremities; their appearance in successive crops; the short duration of each lesion; the absence of subjective phenomena, such as itching and burning; and the presence of rheumatic pains are the diagnostic features.

Pityriasis Rosea.—The eruption is especially found on the trunk; the lesions are rose-red in color; they are slightly scaly, the scales being dry; subjective phenomena are generally absent; and the duration is a few weeks.

Pediculosis Corporis.—Lice may produce a minute red or purple eruption. The small size of the lesions; their confinement to the covered parts; the intense itching and the presence of scratch-marks; and the discovery of pediculi on the clothes are the diagnostic features.

Rötheln.—This affection produces a macular or maculopapular rash that disappears in two or three days by slight desquamation. The moderate fever, sore throat, swollen cervical glands, and history of contagion will assist in the diagnosis.

Accidental Rashes.—Local inflammations like tonsillitis and acute gastritis and certain drugs and foods occasionally produce a macular rash.

Purpuric spots or **hemorrhagic macules** (**petechiæ**) result from minute extravasations of blood into the skin.

A purpuric eruption is observed in the following conditions:

Purpura Hæmorrhagica (*Morbus Maculosus Werlhofii*).—This affection occurs especially in children; it is associated with fever and bleeding from the mucous membranes, and generally runs a course of one or two weeks.

Scurvy.—This disease results from a deprivation of fresh vegetables, and is associated with spongy, bleeding gums, great weakness, and a brawny induration of the muscles.

Rheumatism.—Occasionally an eruption of purpuric spots appears in rheumatic subjects. It is usually associated with pains in the limbs, but fever is generally absent.

Peliosis Rheumatica (*Schönlein's Disease*).—This is an acute affection, characterized by purpuric spots, urticaria, sore throat, moderate fever, and an inflammation of the joints resembling rheumatism. By some the disease is regarded as a manifestation of rheumatism.

Extreme Anemia.—A petechial rash is not uncommon in pernicious anemia, leukocythemia, cancer, and advanced Bright's disease. The history and the associated symptoms of the original disease will indicate the diagnosis.

Certain Infectious Diseases.—In typhus fever a purpuric eruption appears on the fourth or fifth day. In cerebrospinal meningitis the eruption is frequently petechial. In malignant measles and malignant smallpox the rash is often hemorrhagic. In acute yellow atrophy of the liver and in ulcerative endocarditis a petechial eruption is frequently observed.

Poisoning from Certain Substances.—Poisoning from phosphorus, the virus of venomous snakes, mercury, and antipyrin may be associated with an eruption of purpura.

Pediculosis and Kindred Affections.—Body-lice, bedbugs, and fleas produce petechial lesions that are surrounded by slight areolæ. The itching, scratch-marks, and discovery of the parasite are the diagnostic features.

Brown macules are observed in:

Lentigo or Freckle.—The spots are small, and are found especially on exposed parts—face, neck, shoulders, and hands.

Chloasma.—Dark spots may result from irritation of the skin from the action of chemicals, heat, scratches, or blisters. They are sometimes noted in general diseases, like Addison's disease and syphilis. They also occur in primary affections of the skin, as vitiligo, morphea, scleroderma, and leprosy.

Mole or Nævus Pigmentosa.—Moles consist in congenital deposits of pigment on various parts of the body.

White or pale yellow macules are observed in:

Vitiligo.—Apart from the absence of pigment, the skin is normal in appearance and function. An excess of pigment is generally noted at the periphery of the white patches.

Leprosy.—In this condition there are structural changes in the skin and anesthesia in addition to the white appearance.

Morphea.—In the late stage of this affection the circumscribed patches are white or yellow. The structure of the skin is altered, and the periphery of the patches is distinctly hyperemic.

Facial Hemiatrophy.—The onset of this disease may be marked by the appearance of a yellow or white spot on one side of the face.

Diffuse Erythema or Inflammation of the Skin.—Diffuse erythema or inflammation of the skin may result from:

The Action of Certain Drugs (Dermatitis Medicamentosa).—Belladonna, quinin, chloral, cubebs, salicylic acid, and arsenic may produce a diffuse red rash.

Scarlet Fever.—The history of contagion, high fever, sore throat, swollen glands, rapid pulse, and the punctiform character of the rash will indicate the diagnosis.

Rotheln.—In some cases of rötheln the eruption is red and diffuse. The history, slight fever, mild catarrh, and marked swelling of the postcervical glands will suggest rötheln.

Local irritation from traumatism, excessive heat, poisonous plants, or drugs often produces erythema.

Erythema Intertrigo.—This occurs where two cutaneous surfaces come in contact. The part is red, moist, and sometimes macerated. The condition excites a burning pain.

Eczema.—The skin is thickened and infiltrated; there is marked itching; the redness shades off gradually; and there is no fever.

Erysipelas.—The part is considerably swollen; the redness and swelling terminate in an abrupt ridge and the temperature is high.

Acne Rosacea.—This is a chronic disease; the redness appears on the face and is associated with acne lesions and dilated capillaries.

Vesicles.—A vesicle is a small elevation of the skin containing serous fluid, and varying in size from a pinhead to a split-pea. Vesicles are observed in the following conditions:

Sudamen.—This consists of an eruption of minute vesicles that result from the imprisonment of sweat in the layers of the skin. It is usually associated with free perspiration; the vesicles are translucent, lack inflammatory characteristics, and show no tendency to rupture.

Herpes.—The vesicles appear in groups or clusters; they are mounted on an inflammatory base; they show no tendency to rupture; they are frequently associated with burning or neuralgic pains; and they are distributed along the line of the nerve-trunks.

Dermatitis Venenata.—A vesicular eruption may result from contact with poisonous plants, such as the poison-ivy or poison-oak. The eruption generally appears on the exposed parts—face or hands; the part is red and swollen and there is intense itching.

Dermatitis Herpetiformis.—The vesicles are very irregular in shape; they appear in clusters; they are very tense; they show no tendency to rupture; they are frequently associated with other lesions—papules, pustules, and bullæ; they excite intense itching; and they appear in crops over a period of weeks or months.

Impetigo Contagiosa.—The eruption consists of small vesicles that subsequently enlarge until they reach the size of blebs; the vesicles appear in crops; are commonly discrete; are flat and umbilicated; are filled with a straw-colored fluid; they show no tendency to break, but dry up and form thin yellow crusts, and they excite but little itching. The disease is contagious and auto-inoculable; occurs especially in children; and lasts from one to two weeks.

Vesicular Eczema.—The vesicles are quite small and are aggregated in patches; the intervening skin is red and thickened; the vesicles tend to break and pour forth a serous fluid that keeps the part moist; and the eruption is associated with intense itching.

Miliaria, or Heat-rash.—This may appear as an eruption of minute vesicles; they are always discrete; they are surrounded by red areolæ; they usually appear on the trunk; they are generally associated with pin-head papules; they show no tendency to rupture; and they excite a little burning and itching.

Scabies.—In this affection the vesicles are small; they are usually associated with pustules and *burrows;* they excite intense itching; and they are usually found on the hands, forearms, in the axillæ, under the mammæ, and on the inner aspects of the thighs.

Blebs, or Bullæ.—A bleb, or bulla, is a circumscribed elevation of the skin containing serous fluid, and varying in size from a pea to an egg. Blebs are observed in the following conditions:

Impetigo Contagiosa.—The blebs are flat and umbilicated; they contain a straw-colored fluid; they appear in crops; they are commonly discrete; they show no tendency to break, but dry up and form thin yellow crusts; and they excite but little itching. The disease is contagious and auto-inoculable; occurs especially in children; and lasts from one to two weeks.

Dermatitis Herpetiformis.—The bullæ are frequently associated with papules, vesicles, and pustules; they are surrounded by inflamed skin; they appear in clusters; they show no tendency to break, but dry up and leave yellowish-brown crusts; and they excite considerable itching.

Pemphigus.—The bullæ appear in crops; excite but little itching; they lack an inflammatory areola; and, as a rule, they dry up, and leave behind a thin pellicle. The disease is generally chronic.

Syphilis.—The bullous syphilid is observed in hereditary syphilis and very late in the acquired disease. The contents of the bullæ soon become pustular; the blebs dry up, and form dark-green, cone-shaped, stratified crusts, which become detached and leave discharging ulcers. The history and the other evidences of syphilis will aid in the diagnosis.

Pustules.—A pustule is a small circumscribed elevation of the skin containing pus. Pustules are observed in the following diseases:

Eczema Pustulosum.—The pustules are small; are aggregated in a patch; are generally associated with minute vesicles; the intervening skin is red and thickened; and there are marked burning and itching.

Acne Vulgaris.—The pustules are usually confined to the face, back, and shoulders; they have their origin in the sebaceous follicles; they are generally associated with papules and comedones; and they excite no itching.

Dermatitis Herpetiformis.—The pustules are frequently associated with papules and vesicles; they are surrounded by inflamed skin; they appear in clusters; and they excite considerable itching.

Impetigo Contagiosa.—The eruption is at first vesicular, but it soon becomes pustular; the pustules vary in size from a pea to a large marble; they are flat and umbilicated; they appear in crops; they are commonly discrete; they show no tendency to break, but dry up and form thin yellow crusts; and they excite but little itching. The disease is contagious and auto-inoculable; occurs especially in children; and lasts from one to two weeks.

Varicella, or Chicken-pox.—The pustules result from vesicles; they appear especially on the trunk; they are small and are not umbilicated and they excite but little itching.

There is some fever. The disease lasts but three or four days.

Ecthyma.—This disease is observed especially in poorly nourished adults. The pustules vary in size from a pea to a cherry; they are few in number; they are mounted on an inflammatory base, and are surrounded by a distinct inflammatory areola; they excite but little itching; they seldom break, but dry up and form brownish crusts.

Smallpox.—In this disease shot-like papules and umbilicated vesicles precede or are associated with the pustules. The latter are small, surrounded by a red areola, and usually excite some itching. The high fever and history of contagion will assist in making the diagnosis.

Syphilis.—The pustules are frequently associated with other lesions; they are often mounted on a copper-colored inflammatory base; they excite no itching; and they are usually associated with the history and the other evidences of syphilis.

Scabies.—The pustules are small and usually associated with papules, vesicles, and *burrows;* they are especially observed on the hands, forearms, in the axillæ, under the mammæ, and on the inner aspects of the thighs, and they excite considerable itching. There is often a history of contagion.

Papules.—A papule is a circumscribed solid elevation of the skin varying in size from a pin-head to a pea. Papules are observed in the following conditions:

Erythema Multiforme.—The papules are often associated with macules and tubercles; they are flat, and are of a bright-red or purple color; they appear especially on the extremities; and they show no tendency to suppurate, but gradually disappear in the course of two or three weeks; they excite no itching, but they are often associated with prostration and rheumatic pains.

After the Use of Certain Drugs.—Bromids, iodids, copaiba, cubebs, and tar may produce a papular eruption. The history will aid in the diagnosis.

Eczema Papulosum.—The papules are very small, closely aggregated, and often associated with vesicles and pustules; the skin is thickened; and there is intense itching.

Miliaria, or Prickly Heat.—The papules are very small; they are very often associated with minute vesicles; they always remain discrete; they appear especially on the trunk; and they excite a little burning and itching.

Acne Vulgaris.—The papules are usually confined to the face, back, and shoulders; they are generally associated with pustules and comedones; they involve the sebaceous follicles; and they do not excite subjective symptoms.

Scabies.—The papules are small and are usually associated with pustules, vesicles, and *burrows;* they are especially observed on the hands, forearms, in the axillæ, under the mammæ, and on the inner aspects of the thighs; and they excite considerable itching. There is often a history of contagion.

Syphilis.—The papules are dark in color; they are widely distributed, being especially marked on the trunk and flexor surfaces of the extremities; they are usually associated with pustules; and they excite no itching. The history and the accompanying evidences of syphilis will aid materially in establishing the diagnosis.

Smallpox.—The papules are hard and have a shot-like feel; they soon terminate in umbilicated vesicles; they excite some itching, and they are associated with high fever, pain in the back, and often a history of contagion.

Measles.—The papules are small, and run together to form crescentic-shaped patches; and they are associated with moderate fever, swollen cervical glands, coryza, conjunctivitis, and bronchitis. There is often a history of contagion.

Tubercles.—Tubercles are large, circumscribed, solid elevations of the skin varying in size from that of a large pea to that of a walnut. They are observed in the following conditions:

Erythema Nodosum.—The tubercles are large; they usually appear on the extremities; they are reddish-purple in color; they never suppurate; and they are associated with malaise, fever, and rheumatic pains.

Erythema Multiforme.—The tubercles are generally associated with macules and papules; they are flat, and are of a bright-red or purple color; they appear especially on the extremities, and they show no tendency to suppurate, but gradually disappear in the course of two or three weeks. They excite no itching, but are often associated with prostration and rheumatic pains. The disease is probably allied to erythema nodosum.

Lupus Vulgaris.—This may begin as a papule or tubercle. It is especially observed on the face. The tubercles are of a pale-red color and are quite soft to the touch. As a rule, they slowly break down and form shallow ulcers with soft red margins. The ulcers are painless and secrete but little material. They may invade all the soft structures, but the bones escape.

Syphilis.—The tubercular syphilid manifests itself as dark-red tubercles. There are seldom more than three or four, and they generally appear on the face and extremities. They are very firm and often break down, forming deep, punched-out ulcers that secrete an abundant purulent material.

Tinea Sycosis, or Barber's Itch.—The tubercles appear on the hairy parts of the face and involve the hair-follicles. Suppuration soon begins in the center of the tubercles, and the hairs become dry, brittle, and loose. The microscope will reveal the trichophyton.

Leprosy.—One form of leprosy manifests itself as tubercles. The latter are of a pale-red or yellow color, and undergo slow absorption or ulceration. There is usually more or less anesthesia in the parts affected.

Wheals, or Pomphi.—Wheals are evanescent elevations of the skin, generally more or less round, and often white in the center and pale-red at the periphery. They excite considerable itching. They are observed in the following conditions:

Urticaria.—The wheals appear in crops; they are of very short duration; they may appear on any part of the body; and they excite intense itching.

Erythema multiforme, peliosis rheumatica (Schonlein's disease), and certain insects, like mosquitos, also produce wheals.

Crusts.—Crusts consist in dried exudation, and may be red, yellow, brown, or green in color. They are marked in the following diseases:

Eczema.—The crusts are generally associated with pustules and vesicles; the surrounding skin is red and thickened; and there is considerable itching.

Seborrhea.—Crusts of seborrhea are generally observed on the scalp. Itching is absent, and there are no evidences of inflammation.

Syphilis.—The crusts are thick; they are of a dark-brown or green color; and they are often associated with ulcers that freely discharge. The history and other evidences of syphilis will aid in the diagnosis.

Impetigo.—The crusts are thin and yellow, and they are associated with blebs that appear in crops.

Favus.—The crusts generally appear on the scalp; they are yellow, brittle, and cup-shaped; they are usually perforated by a hair, and have a peculiar musty odor.

Tinea Tonsurans, or Ringworm of the Scalp.—In neglected cases this affection may be associated with crusting. It is observed only in children. The grayish scales, the dry, brittle, and broken hairs projecting through the crusts, the alopecia, and the detection of the trichophyton are the diagnostic features.

Scales.—Scales are dry exfoliations from the upper layers of the skin. They are observed in the following diseases:

Squamous Eczema.—The scales are usually associated with papules; the underlying skin is red and thickened, and there is often marked itching.

Seborrhœa Sicca.—The scales are greasy, and the underlying skin shows no evidence of inflammation. The sebaceous follicles are often dilated.

Psoriasis.—The scales are dry, and are of a pearly-white color; they are associated with circumscribed, sharply defined, elevated, inflammatory patches. The extensor surfaces are especially involved. There is little or no itching.

Ichthyosis. — This affection begins in early life. The scales are dry, and are especially marked on the extensor surfaces. Itching is absent, and there is no evidence of inflammation.

Syphilis.—The scales are dry and are of a grayish color; they are usually associated with papules; and they are especially marked on the palms and soles. There is no itching. The history and other evidences of syphilis will assist in the diagnosis.

Pityriasis Rosea.—The scales are found especially on the trunk, and are associated with small, rose-red macules. There is no itching. The disease runs an acute course of a few weeks' duration.

Ringworm.—The scales are dry and scant; they are associated with circumscribed red patches that tend to disappear in the center. There is often marked itching. Microscopic examination reveals the trichophyton.

Ulcers.—Ulcers are observed especially in the following diseases:

Syphilis.—The ulcers are deep; they have a punched-out appearance; they secrete an abundant offensive material; they often involve the bone; they extend rapidly; they are not painful, and the imperfect cicatrix which they produce is soft. The history and other evidences of syphilis will aid in the diagnosis.

Epithelioma.—This appears in late life; there is usually a single center of ulceration; the ulcer is irregular in shape; the edges are thickened and infiltrated; the secretion is scanty and bloody; the progress is somewhat slow, and there is often pain.

Lupus Vulgaris.—This generally appears in early life; there are often several centers of ulceration; the ulcers are usually superficial; the edges are not thickened; the progress is extremely slow; the bones are never involved; there is very little secretion, and soft papules often develop in the cicatrix, which is firm and contracted.

Simple ulcers may result from traumatism, the application of caustics, or the action of intense heat or cold. Ulcers are frequently observed on the legs of old people in association with varicose veins. Simple ulcers may be recognized by the history, location, appearance, and absence of other causes.

Perforating Ulcer of the Foot.—This term is applied to a deep-seated ulcer appearing on the sole of the foot and most frequently observed in locomotor ataxia. It usually begins as a corn in the neighborhood of the great toe, and is generally associated with anesthesia of the sole of the foot.

Decubitus.—This term is applied to the bed-sores that form after the occurrence of grave cerebral or spinal lesions. They are generally observed on parts that are subjected to pressure, as the sacrum, buttocks, calves, and heels, and are preceded by erythema and vesication.

DISEASES OF THE SWEAT-GLANDS.

ANIDROSIS.

Definition.—A deficiency of sweat.

Etiology.—It may be a symptom of some general disease, like diabetes or Bright's disease; it may be an associated condition in certain cutaneous diseases, such as ichthyosis or psoriasis; and it may develop without obvious exciting cause as a result of disturbed innervation.

Treatment.—Remedies should be directed to the primary disease.

HYPERIDROSIS.

Definition.—Excessive sweating.

Etiology.—As a general condition it is often observed in phthisis and in other diseases characterized by marked debility. Local hyperidrosis is most frequently observed in the hands, feet, and axillæ, and probably results from some derangement of the sympathetic nervous system. Unilateral sweating of the face may indicate an aneurysm or tumor pressing on the cervical sympathetic.

Symptoms.—The primary symptom is excessive sweating, and this often leads to intertrigo or eczema. Bromidrosis is often associated with the hyperidrosis.

Prognosis.—Guarded. In many cases the condition is very obstinate.

Treatment.—Frequently there is an evident impairment of the general health that will require appropriate treatment. Internally, one of the following remedies may be employed to diminish the amount of sweat: belladonna, picrotoxin, agaricin, or ergot.

Local Treatment.—Dusting-powders of starch, talc, or lycopodium with tannoform or boric or salicylic acid; or lotions containing sulphate of zinc, tannic acid, or alum are often very useful.

℞. Pulveris acidi salicylici
Pulveris zinci carbonatis præcipitati
Pulveris magnesii ustæ . . . . . āā ʒiv
Pulveris amyli . . . . ʒxv
Pulveris talci . . . . ʒxx.—M.
SIG.—Dusting-powder. (HARDAWAY.)

In hyperidrosis of the feet the method suggested by Hebra is often very efficient. The feet should be washed, thoroughly dried, and then carefully enveloped in strips of muslin that have been spread with diachylon ointment. The application should be made twice daily. In the dressing no water should be employed, but the feet must be carefully wiped and then dusted with starch or lycopodium before the ointment is reapplied. The treatment should be continued for from one to two weeks, after which the feet may be washed and the dusting-powder alone used.

BROMIDROSIS.

(Osmidrosis.)

Definition.—A functional affection characterized by the excretion of sweat that has a fetid odor.

Symptoms.—It is generally local and often confined to the feet; it is frequently associated with hyperidrosis.

Treatment.—Same as hyperidrosis.

CHROMIDROSIS.

Definition.—A functional affection characterized by the secretion of colored sweat.

Symptoms.—The parts most frequently affected are the face and trunk; the most common colors are red and yellow. It is often associated with hyperidrosis.

SUDAMEN.

Definition.—A cutaneous affection, characterized by the eruption of minute vesicles, resulting from the retention of sweat in the upper layers of the skin.

Etiology.—It is often observed in health in persons who perspire freely. It is frequently noted in febrile diseases that are associated with sweating, like pneumonia and typhoid fever.

Symptoms.—Minute, irregular, translucent vesicles appear on the surface. They are not surrounded by an inflammatory areola. They do not rupture, but dry up and are followed by slight desquamation.

Treatment.—The affection has little significance, and treatment is rarely required.

FUNCTIONAL DISEASES OF THE SEBACEOUS GLANDS.

SEBORRHEA.

(**Steorrhea.**)

Definition.—A functional affection, characterized by excessive secretion of sebaceous material, which may be normal or perverted.

Etiology.—In many cases the cause is not apparent. Often the disease is associated with impairment of the general health. By some it is regarded as of parasitic origin.

Varieties.—Seborrhœa sicca and seborrhœa oleosa.

Seborrhœa Sicca.—This form is most frequently observed on the scalp, and constitutes what is popularly termed *dandruff*. Examination reveals an incrustation composed of thin, yellowish-gray, greasy scales. In uncomplicated cases the skin is pale, but from irritation it may subsequently become hyperemic or inflamed. When allowed to continue, the nutrition of the hair is interfered with and baldness results.

On the body seborrhœa sicca appears as yellowish-gray, slightly elevated patches covered with greasy scales. The outlets of the follicles are often dilated. There is generally more or less redness of the skin from hyperemia (*seborrheal eczema*).

Seborrhœa Oleosa.—This form is most commonly observed on the face, particularly about the nose, which is habitually bathed in an oleaginous material that has exuded from the sebaceous follicles. From irritation the parts are often red. The condition is frequently associated with seborrhœa sicca, comedo, and acne.

Diagnosis.—**Eczema.**—In this disease the skin is red and thickened; there is marked itching; and the scales are not greasy.

Psoriasis.—In this disease the scales are dry and pearly and there are evidences of inflammation.

Prognosis.—Favorable under prolonged and judicious treatment.

Treatment.—The general health may be impaired; hence tonics, like iron, strychnin, and cod-liver oil, are often indicated. The gastro-intestinal tract will often require especial attention. Constipation should be relieved by diet, enemas, or mild laxatives.

Local Treatment.—Crusts should be removed by applications of oil, followed by shampooing with alcohol and green soap. When the scalp is thoroughly clean, one of the following remedies may be applied: sulphur, mercury, salicylic acid, carbolic acid, or resorcin.

℞.	Ceræ albæ	℥ij
	Petrolati liquidi	f℥ij
	Aquæ rosæ	fʒvij
	Sodii boratis	gr. x
	Sulphuris	ʒij.—M.

Fiat unguentum.

SIG.—Apply at bedtime for several nights, then shampoo.

Or:

℞.	Resorcinolis	ʒij
	Olei ricini	♏xx
	Spiritus myrciæ	
	Alcoholis	aa f℥iij.—M.

SIG.—Fill an eye-dropper, introduce between the hairs, and subsequently rub in by means of a flannel rag.

Mild cases of facial seborrhea often yield to the following ointment:

℞.	Hydrargyri ammoniati	gr. xx–xxx
	Unguenti aquæ rosæ	℥j.—M.

SIG.—Apply at bedtime.

COMEDO.

Definition.—A functional disease of the sebaceous glands, characterized by the retention of discolored sebaceous material in the distended ducts of the gland.

Etiology.—It is most frequently observed in young adults. Debility, gastro-intestinal disorders, anemia, and lack of cleanliness are predisposing factors.

Pathology.—The material in the ducts is composed of sebum, altered epithelium, and pigment matter that is probably derived from without. Microscopic examination of the material often reveals a mite,—the *Demodex folliculorum*,—but its presence is accidental and of no etiologic significance. Comedo is generally associated with seborrhea.

Symptoms.—The disease is characterized by an aggregation of minute black or yellowish spots that correspond to the outlets of the sebaceous glands. The lesion is often slightly elevated, and when the skin is squeezed, a white, filiform mass exudes, to which the term "flesh-worm" has been popularly applied. The parts most commonly affected are the face, back, and ears. The condition frequently excites an inflammation of the follicles, hence it is often associated with acne.

Prognosis.—Favorable under persistent and judicious treatment.

Treatment.—Anemia, dyspepsia, and constipation must be treated by a careful regulation of the personal hygiene and by the use of appropriate remedies. Tonics, like iron, quinin, cod-liver oil, and strychnin, are often indicated.

Local Treatment.—Large plugs may be pressed out by means of a watch-key or a special instrument for the purpose. Softening and removal of smaller plugs may be hastened by the application of cloths wrung out in very hot water. Kneading and the application of alcohol and green soap will also assist in their expulsion. Mercury and sulphur are useful remedies.

℞.	Hydrargyri chloridi corrosivi	gr. iv
	Alcoholis	f℥j
	Aquæ rosæ	q. s. ad f℥iv.—M.

SIG.—Dab on twice daily.

MILIUM.

(Grutum.)

Definition.—An affection characterized by the appearance of small, pearly, non-inflammatory elevations, which result from the accumulation of inspissated sebum in ducts, the outlets of which have been occluded.

Symptoms.—It is generally observed about the face, and consists of a collection of small, round, pearly elevations, which vary in size from a pin-head to a millet seed. The contents of the distended duct cannot be squeezed out until an opening is made, and thus it differs from comedo. It is frequently associated with comedo and acne.

Treatment.—Mild sulphur ointments are sometimes useful. In obstinate cases the lesions should be punctured, the contents squeezed out, and the interior touched with tincture of iodin.

STEATOMA.

(Wen.)

Definition.—A steatoma, or wen, is a cyst resulting from the retention of secretion in a sebaceous gland.

Symptoms.—One or more rounded or oval elevations, varying in size from a pea to a large walnut, slowly appear on the scalp, face, or back. They are painless, rather soft, and when opened, are found to contain a yellowish-white, caseous mass.

Diagnosis.—**Fatty Tumors.**—Fatty tumors are rare on the scalp; they are frequently lobulated; they have a doughy feel; and are not so movable as wens.

Treatment.—The sac and its contents should be carefully dissected out. Simple excision and evacuation are always followed by a return of the cyst.

INFLAMMATORY DISEASES OF THE SKIN.

ERYTHEMA SIMPLEX.

Definition.—Active hyperemia of the skin.

Etiology.—It may result from exposure to heat or cold; from traumatism; or from the application of some irritating substance. A symptomatic variety is frequently observed in gastric irritation and systemic diseases.

Symptoms.—Diffuse uniform redness, disappearing on pressure, and without thickening or elevation of the skin. When it is marked, there may be slight burning.

Treatment.—Sedative lotions or dusting-powders suffice.

ERYTHEMA INTERTRIGO.

(**Chafing.**)

Definition.—Hyperemia induced by the attrition of opposing surfaces of the skin.

Etiology.—It is common in children and in fat subjects. It is especially noted where there are friction and perspiration, as under pendulous mammæ, between the upper parts of the thighs, and around the genitalia.

Symptoms.—It is characterized by diffuse redness, and often by heat and moisture. It excites a burning sensation. When the cause is continued, it may result in dermatitis.

Treatment.—After bathing the parts with a lotion of boric acid, the following dusting-powder may be used:

℞. Pulveris camphoræ . . . . ʒj
Pulveris amyli
Pulveris zinci oxidi . . . . āā ℥ss.

ERYTHEMA NODOSUM.

(Dermatitis Contusiformis.)

Definition.—An acute inflammatory disease, characterized by crops of large, bright-red nodes that, in the process of evolution, assume different colors, as in the fading of a bruise.

Etiology.—It is usually seen in children. It is frequently associated with rheumatic and digestive disturbances.

Symptoms.—There is a sudden eruption of bright-red nodes, varying in size from a pea to an egg. The extremities are most commonly affected. The advent is marked by malaise, headache, slight fever, and rheumatoid pains. At first the lesions resemble boils, but, unlike the latter, they do not suppurate, but gradually turn yellow, blue, and green, as a bruise.

Prognosis.—Favorable. Duration, a few weeks.

Treatment.—Saline laxatives and sodium salicylate are recommended. Locally, a lotion of lead-water and laudanum makes a soothing application.

ERYTHEMA MULTIFORME.

Definition.—An inflammatory disease characterized by erythematous, papular, vesicular, or bullous lesions.

Etiology.—It is more common in women than in men. It is apt to develop in the spring or fall. Rheumatism and gastro-intestinal disturbances seem to predispose.

Symptoms.—It is marked by an eruption, usually on the extremities, of the following lesions: macules, papules, vesicles, or bullæ. The lesions may aggregate or remain discrete; they last one or two weeks and gradually fade. There is little or no itching. In some cases there is decided constitutional disturbance, manifested by malaise, headache, slight fever, and rheumatic pains.

Diagnosis. — **Dermatitis Herpetiformis.** — The marked itching, the greater tendency for the lesions to cluster, and the chronic character of dermatitis herpetiformis will usually prevent an error in diagnosis.

Urticaria.—In this disease the individual lesions last a very short time and are associated with marked itching.

Prognosis.—Favorable. Duration, a few weeks.

Treatment.—In the debilitated iron and quinin are useful. In the rheumatic, the salts of lithium and of potassium may be employed. Constipation should be relieved by saline laxatives. Locally, lotions of boric or carbolic acid followed by dusting-powders exert a beneficial effect.

URTICARIA.

(Hives; Nettlerash.)

Definition.—An inflammatory affection characterized by the eruption of pale-red, evanescent wheals that are associated with severe itching.

Etiology.—Gastro-intestinal disturbances, emotional excitement, and chronic visceral diseases predispose. In some it may be excited by certain articles of food, such as shellfish, strawberries, etc. The bites of certain insects produce the disease, such as mosquitos, bedbugs, and caterpillars. Some drugs induce urticaria in susceptible people.

Pathology.—The disease consists in a vasomotor spasm, followed by paresis of vessels and an outpouring of serum.

Symptoms.—There is a sudden general eruption of papules or wheals that is associated with intense itching. Each lesion lasts a few hours and is succeeded by new ones in other places.

Varieties.—**Urticaria Papulosa.**—In this form the wheal is followed by a lingering papule that is attended by considerable itching. It is most commonly observed in children.

Urticaria Hæmorrhagica.—The lesions are infiltrated with blood.

Urticaria Tuberosa (Giant Urticaria).—In this form the wheals may reach the size of an egg.

Diagnosis.—**Erythema Multiforme and Erythema Nodosum.**—In both of these affections the lesions last much longer and are free from itching.

Prognosis.—Favorable. In some cases it tends to become chronic.

Treatment.—The cause should be removed when possible. In gastric irritation bismuth or calomel and soda are useful.

When there is constipation, a saline laxative may prove very efficient. The special remedies usually recommended are alkalis, sodium salicylate, quinin, potassium bromid, and atropin.

Locally, lotions of water and alcohol, carbolic acid, boric acid, or hydrocyanic acid are very useful:

℞.	Acidi carbolici	fʒj
	Glycerini	f℥ss
	Alcoholis	f℥j
	Aquæ	q. s. ad f℥viij.—M.

URTICARIA PIGMENTOSA.

This is a form of urticaria observed in young children. It is characterized by an eruption of wheals that are itchy and persistent, and that leave behind a yellowish or brownish pigmentation. The disease runs a chronic course of months or years.

HERPES SIMPLEX.

(Fever-blisters.)

Definition.—An acute, non-contagious disease, characterized by groups of small vesicles mounted on inflammatory bases.

Etiology.—Herpes is very common in febrile diseases, especially pneumonia, influenza, malaria, and cerebrospinal meningitis. Local irritation also predisposes to it. It is dependent upon a peripheral toxic neuritis.

Symptoms.—One or more clusters of small vesicles appear, usually on the face or genitalia. The vesicles are mounted on an inflammatory base, contain clear fluid, and show no tendency to rupture. Soon their contents become puriform, dry up, and form reddish-brown crusts that fall off in a few days. Burning and tingling precede and accompany the eruption.

Varieties.—When it appears on the face, it is termed *herpes facialis;* on the genitals, *herpes progenitalis.*

Diagnosis.—Herpes progenitalis must be distinguished from **chancroid.** The history, the superficial character of the lesion, the burning pain, and the subsequent course will indicate herpes.

Treatment.—The lesion may be painted with flexible collodion, or the following lotion employed:

℞. Zinci oxidi	gr. xv
Glycerini	♏ xv
Liquoris plumbi subacetatis dilutus	♏ x
Liquoris calcis	fʒvj–f℥j.—M.

SIG.—Apply locally. (TILBURY FOX.)

HERPES ZOSTER.

(**Zona; Shingles.**)

Definition.—An acute inflammatory disease, characterized by groups of small vesicles mounted on inflammatory bases, associated with neuralgic pain, and following the distribution of certain nerve-trunks.

Etiology.—The disease commonly depends upon a peripheral neuritis. Injury, exposure to cold, and damp clothes predispose to it.

Symptoms.—Clusters of vesicles mounted on inflammatory bases may appear on any part of the body; but they are most frequently observed along the course of the intercostal nerves. Only one side is affected. Sharp neuralgic pain precedes and accompanies the eruption. The fluid in the vesicles soon becomes turbid, dries up, and forms yellow-brown crusts which fall off in a few days.

Prognosis.—Favorable.

Treatment.—Tonics are often indicated. Bulkley recommends phosphid of zinc in doses of $\frac{1}{8}$ of a grain every three hours. Morphin is sometimes required for the relief of pain. Phenacetin, however, usually suffices.

Locally.—Sedative applications are required; the best are flexible collodion with morphin, or a solution of menthol or carbolic acid, followed by a dusting-powder of oxid of zinc or starch.

℞. Morphinæ sulphatis . . . . gr. ij
Pulveris amyli
Pulveris zinci oxidi . . . . āā ℥ss.—M.

HERPES IRIS.

Definition.—An inflammatory disease, characterized by groups of vesicles arranged in concentric rings that present a somewhat variegated appearance.

Etiology.—The causes are unknown. The disease is rare.

Symptoms.—One or more rings of vesicles successively appear around a central vesicle or papule. The different ages of the rings that compose the patch impart to the latter a variegated appearance. Burning and itching are often attendant symptoms. The hands, arms, and feet are the parts most frequently affected. The lesions appear in successive crops over a period of several weeks. In some instances the vesicles are quite large and resemble the blebs of pemphigus.

Prognosis.—Favorable, but recurrent attacks are common.

Treatment.—The same as in herpes zoster.

ACNE.

(Acne Vulgaris.)

Definition.—An inflammatory disease of the sebaceous glands, characterized by papules and pustules and usually seated on the face or back.

Etiology.—It generally develops about puberty. Anemia, menstrual disorders, and gastro-intestinal disturbances predispose. Certain drugs, like iodid and bromid of potassium and copaiba, may induce the disease.

Pathology.—Acne lesions result from the irritation excited by retained sebaceous matter, hence the papules and pustules are commonly associated with blackheads, or comedones.

Symptoms.—An aggregation of small papules, pustules, and comedones about the face, chest, and shoulders. Pustules or papules predominate according as the disease is acute or chronic. New lesions develop as the old disappear, so that the disease usually runs a protracted course. Subjective phenomena are absent.

Varieties.—**Acne Papulosa.**—In this form the lesion reaches the papular stage and advances no further.

Acne Pustulosa.—In this variety the papules develop into pustules.

Acne Indurata.—The inflammation is deeply seated, the base of the papule or pustule is firm, and the lesion is sluggish.

Acne Atrophica.—In this form the lesions are followed by small scars or pits.

Acne Hypertrophica.—In this form there is an overgrowth of connective tissue and the skin becomes thickened.

Diagnosis.—The distribution, the chronic character of the affection, the involvement of the sebaceous glands, and the association with comedones are the diagnostic features which separate acne from all other affections.

Prognosis.—Curable under persistent treatment.

Treatment.—The general health must be improved. The diet should be nutritious, but easily assimilable; rich food must be prohibited. Constipation should be relieved by mild laxatives. In the anemic and debilitated, iron, quinin, strychnin, and cod-liver oil are useful remedies. The special drugs which have been recommended are arsenic, sulphur, ergot, and calx sulphurata. Arsenic is best suited to the sluggish indurated forms; and calx sulphurata ($\frac{1}{10}$ to $\frac{1}{6}$ grain four times daily) to the pustular variety.

Local Treatment.—In the acute form mild applications should be employed, like the following calamin lotion:

℞.	Pulveris zinci oxidi	ʒij
	Pulveris calaminæ	ʒij
	Glycerini	fʒj
	Aquæ calcis	f℥vj.—M.

In chronic cases the sebaceous plugs should be removed by a watch-key and the pustules incised. Thorough washing with very hot water and green soap is also advisable. The best local remedies are sulphur, mercury, and resorcin.

℞. Calcis	℥ss
Sulphuris sublimati	℥j
Aquæ	f℥x.—M.

Boil together with constant stirring until the mixture measures six ounces and then filter.

Sig.—Apply at first well diluted and gradually increase the strength. (Vleminckx.)

Or:

℞. Sulphuris præcipitatis	ʒj
Unguenti aquæ rosæ	
Petrolati liquidi	āā ʒiv.—M.

Sig.—Apply night and morning. (Van Harlingen.)

Or:

℞. Hydrargyri ammoniati	gr. xx–xl
Unguenti aquæ rosæ	℥j.—M.

Sig.—Use night and morning.

Or:

℞. Hydrargyri chloridi corrosivi	gr. ss–ij
Tincturæ benzoini compositæ	fʒj
Emulsi amygdalæ amaræ	f℥iv.—M.

Sig.—Use locally.

ACNE ROSACEA.

Definition.—A chronic affection, usually located on the face in the region of the nose, and characterized by marked hyperemia, dilatation of the vessels, overgrowth of tissue, and acne lesions.

Etiology.—Anemia, menstrual disorders, gastric disturbances, exposure to extremes of temperature, and intemperance are the usual predisposing causes.

Symptoms.—The affected area is of a deep-red color; the vessels are dilated; the skin is thickened and lumpy, and acne lesions coexist. In advanced cases the nose may become extremely large and lobulated (rhinophyma).

Subjective phenomena are generally absent.

Diagnosis.—**Lupus Vulgaris.**—In this disease there are soft, pale-red papules, ulceration, cicatrization, and no enlargement of the blood-vessels.

Prognosis.—Unless the hypertrophy is marked, the disease is curable under protracted treatment.

Treatment.—The general treatment is the same as in acne vulgaris.

Local Treatment.—Sulphur and mercury are the most reliable remedies. Vleminckx's solution is very useful (see p. 491). Dilated vessels should be destroyed by electrolysis. Large hypertrophies may be removed by the knife.

FURUNCULUS.

(Boil.)

Definition.—An acute circumscribed inflammation of a sebaceous gland or hair-follicle, usually terminating in suppuration.

Etiology.—Single boils are generally due to local irritation. Their appearance in crops (furunculosis) is mainly indicative of impaired health. The entrance of pus cocci into the skin is always essential to their production.

Diagnosis.—Furuncles must be distinguished from **carbuncles**; the latter are single, large, flattened at their summits, and have multiple openings.

Treatment.—In furunculosis the cause should be searched for and, if possible, removed. Tonics like iron, quinin, cod-liver oil, and hypophosphites are often very useful. Calx sulphurata ($\frac{1}{10}$–$\frac{1}{6}$ grain thrice daily after meals) sometimes proves serviceable. A solution of boric acid or of corrosive sublimate may be applied locally. The following paste will sometimes abort them:

℞. Ichthyol . . . . gr. x
Unguenti hydrargyri
Extracti belladonnæ . . . . āā ʒj.—M.
Sig.—Apply locally and make pressure with strips of adhesive plaster.

CARBUNCULUS.

(Anthrax.)

Definition.—A circumscribed inflammation of the skin and deeper tissues, characterized by a dark-red, painful node that breaks down and evacuates through several apertures.

Etiology.—Lowered vitality from any cause predisposes. They are especially common in diabetes. The exciting cause is a special microbe.

Symptoms.—A dark-red, painful, flattened node appears, surrounded by a dusky-red area of induration. In a week or ten days suppuration begins, and the contents are discharged through several orifices. There is generally marked constitutional disturbance. The most common seats are the nape of the neck, back, and buttocks.

Prognosis.—Guardedly favorable. Death is not an infrequent termination in the old and debilitated.

Treatment.—Generally tonics, like quinin, iron, and whisky, are indicated. Opium may be required to relieve pain.

Local Treatment.—In the early stage they may be aborted by a central injection of 10 to 20 minims of a 5 or 10 per cent. solution of carbolic acid in glycerin. When not seen until abortion is too late, firm compression may be made by straps applied concentrically, leaving the central orifice free for the discharge of sloughs; an antiseptic dressing may be applied over the straps. If septicemic symptoms appear, recourse must be had to deep crucial incisions or extirpation of the necrotic tissue.

PSORIASIS.

Definition.—A chronic inflammatory disease, characterized by red, scaly, sharply circumscribed, elevated lesions.

Etiology.—Psoriasis usually develops in young adults. Heredity, the gouty diathesis, pregnancy, and lactation seem to predispose. It is as common in the robust as in the debilitated. It is non-contagious.

Pathology.—The lesions consist of a marked hyperplasia of the rete mucosum, thickening of the horny layer, and round-cell infiltration of the corium.

Symptoms.—Little red spots appear on the body and gradually grow until they reach the size of a dollar. The lesions are of a dull pink or red color, sharply defined, somewhat elevated, surrounded by healthy skin, and covered with abundant dry, pearly, overlapping scales. These scales are readily detached, leaving behind a dry, slightly excoriated surface. The lesions may be uniformly distributed over the entire body, but usually the extensor surfaces are more affected; a symmetric arrangement is often observed. Itching is slightly or entirely absent. After a variable time the center of the patch disappears and leaves behind a spot of healthy skin that gradually increases until no trace of the lesion remains. The disease runs a protracted course of months or years, improving in the summer and growing worse in the winter.

Diagnosis.—**Eczema.**—In this disease the patches are not sharply defined, but shade off gradually into the surrounding skin; there is marked itching; there is usually a decided discharge, and healing begins at the periphery instead of at the center as in psoriasis.

Seborrhea.—In this affection the lesions are usually confined to the scalp and face, while psoriasis is general; the scales are gray and greasy; the patches are not circumscribed and lack the inflammatory character of psoriasis.

Papulosquamous Syphiloderm.—The history, the associated symptoms of syphilis, the coppery color of the lesions, the scant scaling, the special tendency to involve the hands and soles will render the diagnosis apparent.

Prognosis.—The disease disappears under treatment, but relapse generally follows after a longer or shorter period.

Treatment.—The general health may require attention. In the gouty, alkalis are of value; and in the anemic, iron and cod-liver oil are indicated. Arsenic is often of considerable value; it should be given in small doses cautiously increased. Iodid of potassium (10 to 20 grains thrice daily) is sometimes recommended.

Local Treatment.—The scales should be removed by alkaline baths before local applications are made. The best local remedies are tar, oil of cade, chrysarobin, salicylic acid, resorcin, sulphur, and ammoniated mercury. Ointments of chrysarobin and mercury must not be used over too great an area.

℞. Olei cadin.	fʒij
Adipis	℥j.—M.

Sɪɢ.—Apply night and morning.

Or:

℞. Chrysarobini	ʒj
Acidi salicylici	gr. x–xx
Ætheris	fʒj
Olei ricini	♏v
Collodii	q. s. ad f℥j.—M.

Sɪɢ.—Apply with a camel's-hair brush and paint over with plain collodion. (Sᴛᴇʟᴡᴀɢᴏɴ.)

ECZEMA.

(Tetter.)

Definition.—A non-contagious inflammatory disease of the skin, characterized by multiform lesions—erythema, papules, vesicles, pustules, scales, and crusts—and associated with infiltration, itching, and more or less discharge.

Etiology.—It is most common in the young and in the aged. Digestive disturbances, debility, gout, and rheumatism predispose to its development. It may be due to external irritants like cold, heat, the rhus-plant, hard soaps, certain dyes, etc.

Pathology.—The lesions consist of congestion, with a cellular and serous infiltration of the various layers of the skin.

Varieties.—Eczema erythematosum, papulosum, vesiculosum, pustulosum, squamosum, and rubrum.

Eczema Erythematosum.—This form consists in irregular patches marked by swelling, redness, and slight scaling, and accompanied by itching and burning. The most common seat is the face.

Eczema Papulosum.—In this form there is a close aggregation of minute acuminated papules accompanied by severe itching. It is frequently associated with the vesicular variety. The most common seat is the extremities.

Eczema Vesiculosum.—This consists in an ill-defined red patch surmounted by minute vesicles, and accompanied by intense itching. The vesicles soon rupture and leave a raw, weeping surface that becomes more or less covered with crusts. In children it is most common on the face; in adults, on the extremities.

Eczema Pustulosum (Eczema Impetiginosum).—This consists in an aggregation of small pustules that break and lead to the formation of thick yellowish crusts. Itching is not marked. It is frequently associated with the vesicular variety. It is most commonly observed on the face and scalp of poorly nourished children.

Eczema Squamosum.—In this form there are irregular ill-defined red patches accompanied by considerable scaling. It differs from the erythematous form in the large amount of scaling. Its most common seat is the scalp.

When there is a marked tendency to fissuring, as in *chapping*, this form is termed *eczema fissum;* and when there is a tendency to the formation of warty excrescences, it is termed *eczema verrucosum.*

Eczema Rubrum (Eczema Madidans).—This is a secondary variety and is recognized by a raw, dark-red, moist surface, more or less covered with thick, yellowish-red crusts. The itching may be severe. In children it is frequently noted on the face, and in old people on the extremities.

Diagnosis.—**Scabies.**—The history of contagion; the location of the lesions—between the fingers, on the wrists, under the mammæ, in the axillæ; and the presence of burrows will indicate scabies.

Psoriasis.—The sharply defined patches, the dry scaling, the absence of marked itching, the symmetric distribution, and the predilection for extensor surfaces will indicate psoriasis.

Acne Rosacea.—The presence of acne papules and pustules and of dilated blood-vessels and the absence of itching will distinguish acne rosacea from erythematous eczema.

Seborrhea.—The greasy scales and the absence of itching and of all inflammatory symptoms will indicate seborrhea.

Sycosis.—The limitation of the lesions to the hair-follicles of the face and the absence of itching will distinguish sycosis from eczema.

Prognosis.—Generally favorable under persistent and judicious treatment.

Treatment.—*General Treatment.*—The health must be improved. Tonics are frequently indicated. In strumous children cod-liver oil may be of extreme value. Disturbances of the gastro-intestinal tract are frequently present, and will require appropriate treatment. In the gouty and rheumatic the alkaline mineral waters, colchicum, and the salts of lithium are indicated. Constipation must always receive attention. Of the special internal remedies, arsenic is the most important; it is, however, indicated only in the chronic cases in which bright redness, itching, and weeping are absent.

External Treatment.—In acute cases with marked inflammatory symptoms, soothing applications should be employed. A saturated solution of boric acid may be dabbed on for five or ten minutes, and may be followed by zinc ointment spread on lint; when there is much itching, carbolic acid is very useful:

℞.	Acidi carbolici	ʒj
	Glycerini	f℥ij
	Aquæ	q. s. ad f℥viij.—M.

Sig.—Apply locally.

The following is also frequently used:

℞.	Zinci oxidi	℥ss
	Pulveris calaminæ	℈iv
	Glycerini	f℥iss
	Liquoris calcis	f℥viij.—M.

Sig.—Shake and apply locally.

In chronic cases crusts and scales should be removed by soap and water or by:

℞.	Saponis viridis	℥ij
	Alcoholis	℥j.—M.

Sig.—Apply thoroughly and remove with warm water.

The best external applications are those containing salicylic acid, tar, mercury, and resorcin:

℞.	Unguenti picis liquidæ	ʒj–ij
	Unguenti zinci oxidi	q. s. ad ℥j.—M.

Sig.—Apply twice daily.

Or:

℞.	Hydrargyri ammoniati	gr. x–xxx
	Unguenti zinci oxidi	℥j.—M.

LICHEN RUBER, LICHEN PLANUS, AND LICHEN SCROFULOSIS.

Lichen Ruber.—This is an extremely rare disease, characterized by the eruption of small, red, glazed, acuminated papules that show no tendency to coalesce, and that are associated with itching and failure of general health. The disease runs a chronic course, and may prove fatal through exhaustion.

Lichen Planus.—This form is characterized by an eruption on the extremities of small, red, flat papules that tend to spread, and, by coalescing, form dull-red, irregular patches. The lesions have an angular outline, are slightly umbilicated, and at first have a smooth and shiny appearance, but later are slightly scaly. There is more or less itching, but no impairment of the general health. As the old lesions disappear new ones take their place.

Etiology.—These affections are most frequently observed in poorly nourished, middle-aged males.

Treatment.—The general health must be improved by good food and such tonics as iron, strychnin, and cod-liver oil. Arsenic is of considerable value. Locally, ointments of tar or mercury are useful.

Lichen Scrofulosis.—This is a chronic affection occurring chiefly in children of a strumous diathesis, and characterized by small pale-red or salmon-colored scaly papules. They tend to form in groups, and are most frequently observed on the trunk. Itching is absent. The disease runs a chronic course.

Treatment.—Remedies like iron, quinin, and cod-liver oil are indicated. Hebra recommends the last remedy as a local application.

PRURIGO.

Definition.—A chronic inflammatory disease, characterized by a general eruption of minute, discrete papules, accompanied by marked itching.

Etiology.—It is most commonly observed in the poor and ill-fed of Europe. It develops in early childhood and persists through life.

Symptoms.—An eruption of small, discrete, deeply situated, pale-red papules appears on the body, especially on the back and extensor surfaces of the extremities. The skin is harsh, dry, and thickened, and covered with numerous scratch-marks induced by the intense itching.

Prognosis.—Unfavorable; it usually persists through life.

Treatment.—The general health must be improved by good food and the use of nutrient tonics like iron and cod-liver oil. Frequent bathing, followed by ointments of tar, sulphur, or naphthol, gives relief.

DERMATITIS HERPETIFORMIS.

(Herpes Gestationis; Duhring's Disease.)

Definition.—A chronic inflammatory disease, characterized by multiform lesions that form in groups and are associated with intense itching.

Etiology.—Women are more commonly affected than men. Pregnancy, lactation, and menstrual disorders seem to exert a predisposing influence.

Symptoms.—*Erythematous Form.*—This is characterized by the appearance, in crops, of erythematous patches that are associated with considerable itching.

Papular Form.—Groups of papules appear in crops and are frequently associated with erythema, vesicles, and scratch-marks.

Vesicular Form.—Groups of irregularly shaped vesicles resembling herpes appear in crops and are often associated with erythema, pustules, and scratch-marks.

Pustular Form.—This resembles the former, but the vesicles are replaced by pustules.

Bullous Form.—Large, irregularly shaped blebs appear in crops and tend to group. Vesicles and patches of erythema are also frequently present.

Mixed Form.—Vesicles, erythematous patches, pustules, papules, and blebs appear in association, come out in crops, and are attended with intense itching.

In the pustular, bullous, and mixed forms there may be marked constitutional disturbances.

Prognosis.—Guardedly favorable. The disease runs a chronic course. Relapses are very common.

Treatment.—Tonics are generally indicated. Lotions of boric or carbolic acid may be employed to allay itching, and may be followed by a dusting-powder.

DERMATITIS.

Definition.—Inflammation of the skin resulting from the action of some irritant.

Dermatitis Traumatica.—This term is applied to inflammation of the skin resulting from traumatism.

Treatment.—The removal of the cause and the application of soothing remedies will usually suffice.

Dermatitis Venenata.—This term is applied to inflammation of the skin resulting from the application of vegetable, animal, or chemical irritants. Notable examples of this form of dermatitis are observed in susceptible people after exposure to the influence of poison-ivy (*Rhus toxicodendron*), poison-oak (*Rhus venenata*), or poison-sumach (*Rhus diversiloba*).

Symptoms of Rhus-poisoning.—The affection resembles acute eczema, and may appear in a few hours or not until the lapse of several days after exposure to the plant. It is generally observed on the face or hands. The part becomes red and swollen, and soon minute papules and vesicles appear. It gives rise to considerable burning and itching. As a rule, it subsides in a few days, but in patients with sensitive skin it may linger for several weeks.

Treatment.—The part should first be bathed with Castile soap and tepid water, and then treated with some sedative lotion or ointment. Black wash may be dabbed on, and zinc ointment subsequently applied; or a saturated solution of boric acid may be followed by zinc ointment. When there is marked itching, a weak solution of carbolic acid (1 dram to 1 pint) is useful. The fluid extract of grindelia robusta has been highly recommended; it may be applied in the strength of an ounce to a pint of water.

Dermatitis Calorica.—This term is applied to the inflammation of the skin resulting from extreme heat or cold. *Pernio*, or *chilblain*, is characterized by redness, swelling, intense burning, and itching, and results from a sudden change from a low temperature to a high temperature. *Frost-bite* is characterized by congelation; the part is of a dull-white color and is anesthetic; subsequently inflammation or gangrene develops.

Burns and *scalds* result from the application of heat, and are divided into degrees according to the depth to which the destructive process extends.

Treatment.—In *pernio*, or *chilblain*, the part should first be rubbed with snow or bathed in ice-water until the circulation is reëstablished; and then an application made of nitrate of silver (5 grains to the ounce of distilled water) or of tincture of iodin.

In superficial *burns* or *scalds* one of the following remedies may be applied: Phénol sodique, Carron oil (equal parts of linseed oil and lime-water), powdered bicarbonate of sodium, or:

℞. Acidi carbolici . . . . gr. xx
Petrolati . . . . ℥ij.—M.
SIG.—Spread on lint and apply to the wound.

Dermatitis Medicamentosa.—This term is applied to the various cutaneous eruptions that follow the internal use of certain drugs.

Belladonna or Atropin.—These drugs produce a diffuse erythematous rash resembling that of scarlet fever, but it lacks the punctiform character of the latter. It usually appears on the face, neck, and chest, and is associated with dryness of the throat, rapid pulse, and, if the dose has been large, dilated pupils.

Cubebs.—This drug sometimes produces an erythema associated with minute papules.

Copaiba.—The rash may be macular, papular, or like that of urticaria.

Potassium Bromid.—The eruption resembles acne and consists of papules and pustules.

Potassium Iodid.—The eruption may be erythematous, papular, pustular, urticarial, or purpuric. The most common eruption resembles acne, but the lesions are bright-red in color and widely distributed over the surface of the body.

Arsenic.—The eruption may be erythematous, papular, vesicular, or pustular.

Antipyrin.—This drug not infrequently produces a widespread papular eruption.

Quinin.—The rash is usually erythematous, though an urticarial eruption has been observed.

Salicyl Compounds.—The eruption is usually erythematous or urticarial.

Borax.—This drug occasionally produces an eruption resembling psoriasis.

Chloral.—The eruption is usually erythematous or urticarial.

Dermatitis Exfoliativa.—This is a rare affection, characterized by diffuse redness of the skin, high fever and its associated phenomena, and desquamation. It is interesting from its close resemblance to *scarlet fever*, from which it may be distinguished by the history and the absence of sore throat, and a "strawberry" tongue.

ECTHYMA.

Definition.—An inflammatory affection, characterized by the appearance of discrete, flat pustules, which vary in size from a pea to a five-cent piece, and which are surrounded by a distinct red areola.

Etiology.—Male sex, middle life, bad hygiene, and debility are predisposing factors.

Symptoms.—Flat, yellow pustules appear in crops. They are surrounded by a distinct red areola and soon dry up, forming reddish-brown crusts. Slight excoriation and pigmentation sometimes remain after the separation of the crusts. Subjective phenomena are usually absent.

Diagnosis.—The acute course, the absence of ulceration, and the absence of history and of associated symptoms of syphilis will separate it from the **pustular syphilid.**

Impetigo.—In this affection the lesions are not flat; they are not distinctly inflammatory; and the crusts are light yellow, not reddish-brown. Impetigo occurs most frequently in children, who may be quite robust.

Prognosis.—Favorable.

Treatment.—Constitutional treatment is generally required. Such tonics as iron, quinin, strychnin, and cod-liver oil are often indicated.

Local Treatment.—The crusts should be removed and some stimulating ointment applied, as the following:

℞. Hydrargyri ammoniati . . . . gr. x
Unguenti zinci oxidi . . . . ℥j.—M.

PEMPHIGUS.

Definition.—A non-contagious inflammatory disease, characterized by the eruption of successive crops of bullæ or blebs.

Etiology.—Female sex, nervous prostration, heredity, and injury to the peripheral nerves are predisposing factors.

Varieties.—Pemphigus vulgaris and pemphigus foliaceus.

Pemphigus Vulgaris.—This form usually runs a chronic course, and is characterized by successive crops of blebs, varying in size from a small pea to a large walnut. They are thoroughly distended with fluid, which is at first clear, but subsequently turbid. As a rule, they do not rupture, but disappear in the course of five or six days, their contents being gradually absorbed. After absorption a thin pellicle remains, which dries and is subsequently detached, leaving behind a slightly pigmented spot. No part of the body is exempt; and as one set of blebs disappears, new ones rapidly develop, and so the disease continues for many years.

In severe cases there may be considerable itching and burning attending the eruption.

Pemphigus Foliaceus.—This rare and grave form of pemphigus is characterized by crops of blebs, which are flaccid and filled with a turbid fluid almost from the beginning. They soon rupture and form thick crusts, which, separating, leave behind red weeping surfaces. The crops follow each other in rapid succession, and at times the whole body may be covered with blebs and scabs. The disease may last several years, death ultimately resulting from exhaustion.

Diagnosis.—**Bullous Syphiloderm.**—The history, the associated symptoms of syphilis, the thick, yellow, stratified crusts, and the underlying ulceration will serve to separate this affection from pemphigus.

Impetigo Contagiosa.—The acute course, the contagious and auto-inoculable character of the affection, and the umbilication of the blebs will separate impetigo contagiosa from pemphigus.

Prognosis.—The prognosis should be guarded. Pemphigus vulgaris runs a long course and is often intractable. Pemphigus foliaceus often proves fatal through exhaustion.

Treatment.—The diet should be nutritious, but carefully adapted to the stomach. The patient should be placed under the best hygienic conditions. Tonics, like iron, quinin, phosphorus, cod-liver oil, and strychnin, are usually indicated. In many cases arsenic is a valuable remedy.

Local Treatment.—The blebs may be punctured and subsequently dressed with zinc ointment.

IMPETIGO CONTAGIOSA.

Definition.—An acute, contagious, inflammatory disease, characterized by flat, yellowish blebs that dry up and form thin, yellow, lamellated crusts.

Etiology.—Its exciting cause is unknown. It is most frequently observed in debilitated children.

Symptoms.—The eruption is most frequently observed on the face and extremities; it generally appears in crops, and is at first vesicular. The vesicles grow, and are soon converted into flat, umbilicated pustules which vary in size from a pea to a large walnut. They have a slight red areola. Itching is slight or entirely absent. In some cases there is moderate fever with its associated phenomena. In a few days the blebs dry up and form thin, yellow, lamellated crusts that, separating, leave a slightly excoriated surface. The disease is contagious, and the lesions are auto-inoculable.

Diagnosis.—**Eczema.**—In this disease the pustules are deeper, more confluent, excite intense itching, and are associated with inflammation and infiltration of the surrounding skin.

Prognosis.—Favorable. It terminates spontaneously in a few days or weeks.

Treatment.—A slight stimulating ointment like the following is sometimes useful:

℞. Hydrargyri ammoniati . . . . gr. x–xx
Adipis . . . . ℥j.—M.
SIG.—Apply to the surface after removal of the crusts.

MILIARIA.

(Prickly Heat.)

Definition.—A mild inflammatory disease of the sweat-glands, characterized by the occurrence of minute papules and vesicles.

Etiology.—Extreme heat is the principal predisposing cause.

Symptoms.—The eruption generally appears on the trunk, and consists of minute, closely aggregated red papules or clear vesicles. The lesions are discrete, and excite some burning and itching. It is generally associated with free perspiration.

Diagnosis.—**Eczema papulosum** differs from miliaria in that the papules are larger, appear more gradually, disappear more slowly, and excite intense itching.

Eczema vesiculosum differs from miliaria in that the vesicles are large, disappear more slowly, show a tendency to break, and are associated with marked itching.

Sudamen differs from miliaria in that it lacks all inflammatory characteristics.

Prognosis.—Favorable. Obstinate cases may persist for several weeks.

Treatment.—The general health may require attention. The diet should be light and easily assimilable. Constipation should be relieved by saline laxatives. Locally, a lotion of boric acid, followed by a simple dusting-powder, is generally all that is required.

℞.	Pulveris amyli	ʒvj
	Zinci oxidi	ʒiss
	Pulveris camphoræ	ʒss.—M.

SIG.—Dusting-powder. (HARDAWAY.)

ALBINISM.

Definition.—A congenital deficiency of pigment.

Etiology.—Beyond heredity, no cause is known. Partial albinism is more common in the negro.

Symptoms.—In complete albinism the skin is white; the hair is thin, soft, and very light in color; the pupils appear red, the eyes are very sensitive to light, and the iris and choroid are deficient in pigment.

VITILIGO. 2

(**Leukoderma.**)

Definition.—An acquired cutaneous affection, characterized by milk-white patches that are surrounded by areas of increased pigmentation.

Etiology.—The disease seems to be more common in the tropics and in the colored race. The condition probably results from disturbed innervation.

Symptoms.—Milk-white spots appear on the body and grow very slowly; their borders usually reveal an increase of the normal pigment. Apart from the absence of pigment the skin is normal.

Diagnosis.—**Morphea.**—The initial hyperemia and the subsequent atrophy of the skin will serve to distinguish morphea from vitiligo.

Anesthetic Leprosy.—The subjective symptoms, the atrophy of the tissues, and the anesthesia will separate leprosy from vitiligo.

Prognosis.—Unfavorable; the disease usually persists through life.

Treatment.—Tonics and local stimulants may be tried. Among the latter, electricity, blisters, and irritating ointments have been recommended.

CANITIES.

Definition.—Grayness of the hair.

Etiology.—Local grayness may be congenital or result from some disturbance of innervation, as in neuralgia of the supra-orbital nerve. As a general condition it is usually an expression of senility, although it occasionally develops very early in life. Profound emotional disturbances sometimes induce an abrupt development of canities.

Prognosis.—The condition is permanent, and treatment is of no avail.

ATROPHY OF THE SKIN.

Etiology.—Atrophy of the skin occurs under several conditions. A local atrophy may result from inflammation or injury of a nerve-trunk; in these cases the wrinkles are absent, the skin is thin, smooth, and shiny, and there is often intense burning in the part ("*glossy skin*"). Atrophy is also observed in leprosy, morphea, and scleroderma.

Universal atrophy of the skin results from senility, and very rarely as an idiopathic condition. Sometimes the atrophy occurs in lines or spots (*striæ et maculæ atrophicæ*) as an idiopathic condition, or as the result of stretching the skin, as in the *lineæ albicantes* following pregnancy.

ATROPHY OF THE HAIR.

Etiology.—Atrophy of the hair may result from local diseases that interfere with the nutrition of the scalp, such as seborrhea, eczema, ringworm, etc.; or it very rarely arises as an idiopathic condition without obvious cause.

Prognosis.—When the cause can be ascertained and removed, the prognosis is favorable.

Treatment.—Local diseases will require appropriate treatment. The general health should be improved. Stimulating applications of mercury, sulphur, or carbolic acid are sometimes useful.

ATROPHY OF THE NAILS.

Etiology.—Occasionally the condition is congenital, but more frequently it is acquired, and results from injury or disease of the nerve-trunk; from some general disease, like one of the fevers, syphilis, or cancer; or from some disease of the skin, as psoriasis or ringworm.

Symptoms.—The nails lose their luster, cease to grow, and become opaque and brittle.

Prognosis and Treatment.—Both will depend on the exciting cause.

ALOPECIA.

(Baldness.)

Etiology.—(1) Baldness may be congenital; in these cases it is usually partial. (2) It may be an expression of senility, in which case it generally begins on the crown or brow, and is associated with more or less atrophy of the scalp. (3) It may occur early in life, as an idiopathic affection arising without obvious cause. (4) It may result from general diseases, like syphilis and the fevers. (5) In early life it is often due to some local disease, especially seborrhea.

Prognosis.—In congenital, senile, and idiopathic alopecia the prognosis is unfavorable. In the alopecia of general diseases the prognosis is usually favorable. In alopecia resulting from seborrhea much can be accomplished by persistent and judicious treatment.

Treatment.—The general health should be improved. Frequently washing the head with warm water and Castile soap is to be recommended. One of the following local stimulants may be prescribed: Cantharides, quinin, alcohol, capsicum, sulphur, or carbolic acid.

℞.	Tincturæ cantharidis	f℥j
	Acidi carbolici	ʒj
	Olei ricini	ʒiss
	Spiritus myrciæ	
	Spiritus lavandulæ	aa f℥ij.—M.

Or:

℞.

Tincturæ cantharidis	fʒij
Quininæ sulphatis	gr. x
Glycerini	fʒij
Olei rosmarini	gtt. v
Spiritus myrciæ	q. s. ad f℥v.—M.

When there is much dandruff, the following lotion will be found useful:

℞.

Resorcinolis	ʒij
Acidi salicylici	gr. xxx–ʒj
Olei ricini	f℥ss–j
Olei bergamottæ	fʒj
Alcoholis	q. s. ad f℥vj.—M.

(SCHAMBERG.)

ALOPECIA AREATA.

(Alopecia Circumscripta.)

Definition.—Baldness appearing in circumscribed patches without any obvious lesion of the skin.

Etiology.—The cause is unknown. Some regard it as of parasitic origin, while others look upon it as a neurosis. It is generally observed in early adult life.

Symptoms.—The disease is characterized by the sudden or gradual appearance of circumscribed round patches of baldness. At first there is no change in the appearance of the skin, but later it may become pale and atrophied. Although the scalp is the most frequent seat, it occasionally involves other hairy parts, as the eyebrows, beard, etc.

Diagnosis.—**Ringworm.**—Ringworm is exceedingly rare in adults, and is characterized by elevated scaly patches through which project dry, brittle, broken hairs. If there should be any doubt in the diagnosis, the microscope may be employed to detect the trichophyton.

Prognosis.—In the majority of cases the hair returns under prolonged and persistent treatment. The older the patient, the less favorable the prognosis.

Treatment.—General tonics, like iron, arsenic, quinin, and strychnin, are usually indicated. The local treatment should be stimulating and consist in the application of blisters, electricity, friction, rubefacient liniments, or ointments containing chrysarobin, tar, sulphur, or ammoniated mercury.

℞.	Tincturæ cantharidis	
	Tincturæ capsici	aa f℥ss
	Olei ricini	f℥ss
	Spiritus rosmarini	f℥ij
	Alcoholis	f℥j.—M.

Or:

℞.	Betanaphtholis	gr. xl–ʒj
	Petrolati	℥j.—M.

SYCOSIS.

(Simple Sycosis; Folliculitis Barbæ.)

Definition.—A non-contagious inflammatory disease of the hair-follicles.

Etiology.—The affection results from local irritation and the entrance of pyogenic cocci.

Symptoms.—The disease usually manifests itself on the bearded region of the face, and is characterized by an aggregation of papules and pustules, each of which is pierced by a hair. When the lesions are discrete, the intervening skin is often quite healthy; but when they are close together, it is often infiltrated and hyperemic. During the papular stage the hairs are not loose, but firmly attached; during the pustular stage, however, they can be readily extracted. The pustules show no tendency to rupture, but dry to yellowish-brown crusts. Acute cases are associated with more or less burning and itching. If the disease persists, it may lead to extreme destruction of the hair-follicles, and, as a consequence, to permanent alopecia.

Diagnosis.—**Eczema.**—The lesions in eczema excite severe itching, are not perforated by hairs, and are not confined to the hairy parts.

Tinea Sycosis, or Barber's Itch.—The affection begins as a red, scaly patch, and is followed by the development of large, deeply seated tubercles. The hairs soon become dry, brittle, and broken off, and can easily be extracted. In doubtful cases the microscope may be employed for the detection of the trichophyton.

Prognosis.—The disease is curable under prolonged and judicious treatment. Relapses are very prone to occur.

Treatment.—In acute cases soothing applications are indicated; thus the parts may be dabbed with black wash or a saturated solution of boric acid, and subsequently spread with oxid of zinc ointment. In chronic cases the crusts should be removed, and the hairs cut close or, preferably, shaved. It is advisable to puncture the pustules and to extract the hairs, so as to preserve the follicles. When the parts are not irritable, stimulating applications are useful, and one of the following may be selected:

℞. Sulphuris præcipitatis . . . . gr. xxx–ʒiss
Unguenti aquæ rosæ . . . . ℥j.—M.
Sig.—Apply twice daily.

Or:

℞. Ichthyol . . . . ʒj
Petrolati . . . . ℥j.
Sig.—Apply twice daily.

POMPHOLYX.

(Dysidrosis.)

Pompholyx is a very rare disease, usually observed in those who perspire freely, and characterized by an eruption of deeply seated vesicles that resemble sago-grains imbedded in the skin. The vesicles most commonly appear on the hands, especially between the fingers, and gradually increase in size until they reach the dimensions of blebs. They show no tendency to rupture, but dry up, and are followed by extensive desquamation of the cuticle. The eruption often excites considerable pain and tenderness. The disease usually disappears in the course of a few weeks, but is prone to recur.

Treatment.—General tonics, like iron, strychnin, and arsenic, are often indicated. Locally, sedative lotions or ointments should be employed.

LENTIGO.

(Freckle.)

Definition.—A deposition of pigment in the form of small, irregularly shaped brownish spots.

Etiology.—Blondes are more subject to the affection than brunettes. Exposure to the sun's rays often serves as an exciting cause.

Symptoms.—Exposed parts—the face, shoulders, arms, and hands—are mostly affected. The patches vary in color from yellow to dark brown, and range in size from a pin-head to a pea.

Prognosis.—Freckles can be removed, but they always return.

Treatment.—One of the best remedies is the bichlorid of mercury in solution or ointment.

℞. Hydrarg. chlor. corros. . . . . gr. iv–viij
Alcoholis et aquæ . . . . āā q. s. ad f℥iv.—M.
Sig.—Apply twice daily.

CHLOASMA.

Definition.—An abnormal deposition of pigment in the form of large brown or liver-colored patches.

Etiology.—It may result from the application of external irritants; from general diseases like malaria and Addison's disease; or from affections of the uterus, as pregnancy, tumors, etc.

Symptoms.—The affection consists in the appearance—especially on the face—of large, round, or irregularly shaped brownish or blackish patches. Apart from the discoloration the skin is normal.

Diagnosis.—In **leukoderma** the periphery of the patches is pigmented, but the central milk-white appearance is not seen in chloasma.

Prognosis.—When the cause can be removed, the prognosis is favorable.

Treatment.—When possible, the cause should be removed. The best local remedies are bichlorid of mercury and sulphur.

℞.	Hydrargyri chloridi corrosivi	gr. iij–xij
	Acidi acetici diluti	fʒij
	Sodii boratis	gr. xl
	Aquæ rosæ	q. s. ad f℥iv.—M.

SIG.—Apply night and morning. (HARDY.)

KERATOSIS PILARIS.

(Lichen Pilaris.)

Definition.—Small, papular elevations resulting from hypertrophy of the epidermis surrounding the outlets of the hair-follicles.

Etiology.—It generally results from infrequent bathing.

Symptoms.—The skin, particularly on the extensor surfaces of the arms and legs, is the seat of numerous pin-head elevations which have a dirty-gray color and are pierced by hairs. It may excite some itching. Generally there are no evidences of inflammation, but sometimes a few red papules or even pustules result from irritation.

Diagnosis.—In **cutis anserina**, or goose-flesh, the lesions are transient and have the color of normal skin.

Prognosis.—Favorable.

Treatment.—In most cases nothing will be required beyond frequent bathing with soap, followed by friction of the skin. In obstinate cases some simple ointment may be applied after bathing.

MOLLUSCUM EPITHELIALE.

(Molluscum Contagiosum; Molluscum Sebaceum.)

Definition.—A cutaneous affection, characterized by the appearance of discrete, wax-like elevations ranging in size from a pin-head to a pea, and varying in color from white to rose-pink.

Etiology.—The disease is generally observed in children, and frequently affects several members of the same household, school, or asylum. It is probably contagious.

Symptoms.—Small white or pale-pink, wax-like elevations appear, especially on the face. They are always discrete and rarely abundant. The center of the elevation is depressed and reveals a dark spot that corresponds to the aperture of the follicle. At first the lesions are quite firm, but as they grow old they become soft. When firmly squeezed, they exude a soft, cheesy material. After remaining for several weeks they break down or undergo slow absorption.

Diagnosis.—The color, the wax-like appearance, the umbilication, and the central aperture are the diagnostic features.

Prognosis.—Favorable, although the disease may run a protracted course of months or years.

Treatment.—General tonics, like iron, strychnin, and arsenic, are often indicated. The lesions should be incised, the contents expressed, and their bases touched with nitrate of silver; ointments of mercury and sulphur have also been recommended.

CALLOSITAS.

(**Callus; Keratoma; Tylosis.**)

Definition.—A thickened, horny condition of the skin resulting from hypertrophy of the corneous layer of the epidermis.

Etiology.—Constant irritation from friction or pressure is the chief cause; hence it is frequently seen on the feet from the rubbing of shoes, and on the hands from the friction of tools.

Symptoms.—The condition is characterized by the appearance of hard, thickened, grayish masses, which gradually merge into healthy skin. The soles and palms are the parts most frequently affected. When slight, it causes little inconvenience, but occasionally it becomes fissured and painful.

Prognosis.—It yields rapidly to treatment when the cause is removed.

Treatment.—When excessive, the parts should be soaked and the thickened epidermis pared off. One of the best remedies for softening the horny overgrowth is salicylic acid; it may be applied in the form of a plaster or in collodion.

℞. Acidi salicylici	ʒj
Olei ricini	♏x
Collodii	f℥j.—M.

SIG.—Apply night and morning.

CLAVUS.

(Corn.)

Definition.—Clavus is a circumscribed thickening of the epidermis usually appearing on the feet.

Etiology.—Corns generally result from the friction of ill-fitting shoes.

Symptoms.—Small, circumscribed, horny elevations appear upon the feet and often excite severe pain. When bathed in perspiration, they become more or less macerated, and in this condition constitute the so-called *soft corn.*

Treatment.—A radical cure requires the use of well-fitting shoes. The corns may be removed by soaking, paring, and the application of some mild caustic like salicylic acid.

℞. Acidi salicylici	gr. xxx
Tincturæ iodi	♏x
Extracti cannabis indicæ	gr. x
Collodii	f℥ss.—M.

SIG.—Apply night and morning for several days, and then soak in hot water.

CORNU CUTANEUM.

(Cutaneous Horn.)

Definition.—A circumscribed, projecting outgrowth resulting from hypertrophy of the epidermis.

Symptoms.—Horns generally appear on the face, scalp, or penis, and are usually observed in the old. They consist of dry, rough, horny, more or less conic projections, which vary in length from a few lines to several inches.

Prognosis.—Favorable.

Treatment.—The horn should be excised and the base subsequently cauterized.

VERRUCA.

(Wart.)

Definition.—A wart is a circumscribed elevation resulting from hypertrophy of the papillæ and epidermis.

Etiology.—The cause is obscure. A bacterial origin has been suggested. They are most frequently observed in children.

Symptoms.—*Verruca vulgaris*, or common wart, is generally observed on the hands of children. It consists of a firm, circumscribed elevation, varying in size from a millet-seed to a pea.

Verruca plana, or flat wart, is a circumscribed, flat, pigmented elevation usually observed on the backs of old people.

Verruca Filiformis.—This is a thread-like overgrowth, and is generally observed on the soft parts, like the face and neck.

Verruca Digitata.—This form is made up of numerous branches, and is generally observed on the scalp.

Verruca Acuminata, or Venereal Wart.—This appears in groups about the genitalia. It is soft, red in color, and highly vascular. It may be dry or moist, according to its location; the latter condition often gives rise to a peculiarly offensive odor.

Treatment.—Ordinary warts may be removed by excision, caustics, or electrolysis.

Venereal warts should be bathed in some antiseptic solution and subsequently dusted with calomel, iodoform, or boric acid.

NAEVUS PIGMENTOSUS.

(Mole.)

Definition.—A circumscribed deposit of pigment, usually associated with hypertrophy of cutaneous structures.

Etiology.—Moles are usually congenital.

Symptoms.—The neck, face, and trunk are favorite localities. The nevi vary in number from one to several hundred; in size, from a millet-seed to a filbert; and in color, from yellow to black. When the surface is smooth, the growth is termed *nævus spilus;* when the surface is covered with hair, it is termed *nævus pilosus;* when the surface is warty, it is termed *nævus verrucosus;* and when there is much overgrowth of connective tissue, it is termed *nævus lipomatodes.*

Treatment.—They may be removed by excision, the application of caustics, or by electrolysis.

ICHTHYOSIS.

(Fish-skin Disease.)

Definition.—A chronic affection, characterized by dryness, thickening, and scaliness of the epidermis.

Etiology.—The affection is often hereditary, and is usually detected in early childhood.

Symptoms.—The skin is dry and harsh; the surface is covered with adherent polygonal scales; and the papillæ are more or less hypertrophied. The term *ichthyosis hystrix* is applied to the condition when there is excessive hypertrophy of the papillæ. The extensor surfaces of the extremities are the parts most involved.

Diagnosis.—The absence of all inflammatory symptoms will separate ichthyosis from *squamous eczema* and *psoriasis.*

Prognosis.—The disease is incurable, but the patient can be rendered comfortable by appropriate treatment.

Treatment.—The scales may be removed by alkaline baths or by applications of green soap. The skin may be rendered pliable by rubbing in some simple ointment.

℞. Sulphuris	gr. xxv–l
Adipis	℥j.—M.

Sig.—Rub in at night. (Unna.)

ONYCHAUXIS.

Onychauxis, or hypertrophy of the nail, may be congenital, or may result from certain skin affections, such as eczema, ringworm, or syphilis; from diseases of the nerves, as neuritis; or from traumatism.

HYPERTRICHOSIS.

(Hirsuties.)

Hypertrichosis, or hypertrophy of the hair, may be local or general. The term is applied not only to an excessive overgrowth of hair, but to a growth of hair in unusual localities, as on the faces of young women.

Treatment.—The hair may be removed temporarily by shaving, epilation, or depilatories. Permanent relief can be accomplished only by electrolysis.

SCLERODERMA.

(Sclerema; Scleriasis.)

Definition.—A pigmented, rigid, indurated condition of the skin, occurring in circumscribed patches or involving the entire body.

Etiology.—The cause is unknown.

Symptoms.—The affection may be diffuse or involve circumscribed patches. It may appear quite suddenly, or develop very gradually in the course of months or years. The skin assumes a yellowish-brown color, becomes rigid, indurated, and hide-bound; the surface is unnaturally dry and smooth. When the condition is advanced, the joints become more or less immobile.

Prognosis.—Guarded. It often recovers spontaneously after having persisted for a long time. In other cases the process may spread until the patient becomes almost helpless.

Treatment.—Tonics, like iron, arsenic, and cod-liver oil, are often indicated. Locally, massage, friction, electricity, and inunctions are recommended.

MORPHEA.

(Addison's Keloid.)

Definition.—A cutaneous affection, characterized by circumscribed, rounded, ivory-like patches, which have hyperemic or pigmented borders.

Etiology.—The cause is unknown. By many it is regarded as a circumscribed form of scleroderma.

Symptoms.—The lesions usually appear upon the trunk, and consist of sharply circumscribed patches, which are at first slightly hyperemic. The surface is smooth and resistant to the touch. As the patch grows old its center becomes pale and ivory-like, while the periphery remains hyperemic or becomes pigmented.

Prognosis.—Guarded.

Treatment.—The same as scleroderma.

ELEPHANTIASIS.

(Elephantiasis Arabum; Elephantiasis Pachydermia; Barbadoes Leg.)

Definition.—Hypertrophy of the skin and subcutaneous tissues, usually associated with lymphangitis, edema, and pigmentation.

Etiology.—While elephantiasis may occur in any part of the world, it is far more common in the tropics. It is most frequently observed in the male sex, and rarely develops before adult life. It results from obstruction of the lymphatics, and the most common cause of such obstruction is the presence of a parasite—Filaria sanguinis hominis.

Pathology.—Examination of the affected tissues reveals hypertrophy of the connective tissue, edema, and inflammation and dilatation of the lymphatic vessels.

Symptoms.—It usually begins with recurring attacks of erysipelatoid inflammation. The part is red, swollen, and painful; the lymphatics may be traced as branching red lines beneath the skin; and with these local phenomena there is more or less fever. After each attack the part is left a little enlarged, until finally it presents the following characteristic appearance: it is enormously swollen; the skin is thickened, roughened, and pigmented; and the papillæ are unusually prominent. The regions generally affected are the legs and genitals. In elephantiasis of the scrotum (*lymph-scrotum*) the hypertrophied mass may weigh as much as 50 or even 100 pounds.

Prognosis.—In the early stage the disease may be arrested, but when fully established, it is incurable.

Treatment.—The acute inflammatory attacks should be treated by rest and the application of sedative lotions, like lead-water and laudanum. Subsequently mercurial inunctions may be employed, and the part firmly bandaged with the view of promoting absorption. Amputation may be successfully employed in lymph-scrotum. In elephantiasis of the limbs ligation of the main artery has given somewhat encouraging success.

DERMATOLYSIS.

(Pachydermatocele; Cutis Pendula.)

Definition.—A circumscribed hypertrophy of the skin and subcutaneous tissues resulting in a softened and pendulous condition of the integument.

Symptoms.—The part affected is thickened and pigmented; it is soft and fat-like to the touch; and when the condition is marked, the skin hangs in folds. The regions generally affected are the shoulders, arms, back, and buttocks.

Treatment.—The redundant tissue may be removed by excision or electrolysis.

KELOID.

(Cheloid; Kelis.)

Definition.—A new growth resulting from hypertrophy of the connective tissue of the corium.

Etiology.—It generally results from local injury, though it is claimed that it may arise spontaneously. Certain families and individuals are especially predisposed. It is more frequent in the colored race.

Symptoms.—It begins as a pale-red nodule, which slowly increases in size and sends out claw-like processes. From its resemblance to a crab it has been termed keloid. It is firm, elastic, slightly elevated, sharply defined, and ranges in size from a small bean to a growth as large as the hand. It sometimes excites pain and itching, but generally subjective phenomena are absent. The regions most frequently involved are the chest and back.

Diagnosis.—Keloid may be distinguished from a **hypertrophied scar** by the fact that the latter does not extend beyond the limits of the injury.

Prognosis.—The growth is usually permanent, and after removal invariably returns.

Treatment.—It may be removed temporarily by excision, electrolysis, or caustic pastes.

FIBROMA.

(Molluscum Fibrosum.)

Definition.—A circumscribed overgrowth derived from the subcutaneous connective tissue.

Etiology.—Early life and heredity are predisposing factors.

Symptoms.—The tumors are circumscribed; painless; soft or firm; often multiple; range in size from a pea to a hen's egg; and do not impair the general health. The overlying skin may be normal in appearance or slightly hyperemic.

Prognosis.—They are permanent and treatment is rarely indicated.

ANGIOMA.

(Nævus Vasculosus.)

Definition.—A new growth, composed of cavernous tissue or a congeries of small blood-vessels.

Angioma Cavernosum.—This form is congenital, is composed of cavernous tissue, and appears as a circumscribed, elevated, dark-red tumor that ranges in size from a pea to one as large as the hand. It is often lobulated and pulsating.

Angioma Simplex (**Capillary Nevus; Port-wine Mark**).—This form is also congenital, and is composed of a congeries of capillaries. It is non-elevated, bright-red or purple-red in color, and may cover an area of several inches. It is generally found on the face, and constitutes what is popularly termed a *mother's mark.*

Telangiectasis.—This form is acquired, and is composed of dilated or newly developed capillaries. It appears as a bright-red dot from which branch dilated capillaries. It is frequently associated with acne rosacea; it is also common in those of a gouty diathesis and in those much exposed to the weather.

Treatment.—Cavernous angiomata may be removed by ligation, excision, or electrolysis. Simple angiomata and telangiectasis are best treated by electrolysis.

XANTHOMA.

(Vitiligoidea; Xanthelasma.)

Definition.—A circumscribed connective-tissue new-growth appearing as flat patches or tubercles of a yellowish color.

Etiology.—Middle life and female sex are general predisposing factors. Hepatic disorders, especially obstructive jaundice, seem to exert a decided predisposing influence.

Symptoms.—There are two forms: *xanthoma planum*, which generally appears about the eyelids and consists of smooth, circumscribed, slightly elevated, buff-colored patches; and *xanthoma tuberosum*, which may appear on the neck, shoulders, trunk, or extremities, and consists of small, elastic, and yellowish-colored nodules.

Treatment.—These growths may be removed by excision, electrolysis, or caustics.

LUPUS ERYTHEMATOSUS.

(Seborrhœa Congestiva.)

Definition.—Lupus erythematosus is a new-growth resulting from a cellular infiltration of the skin, and characterized by circumscribed, red patches that are more or less covered with yellowish-gray adherent scales.

Etiology.—Middle life and female sex are predisposing factors. It frequently arises from disorders of the sebaceous glands, as seborrhea or acne.

Pathology.—By many it is regarded as a chronic dermatitis which originates in the sebaceous glands.

Symptoms.—The disease usually manifests itself on the face, in the region of the nose, and appears as small, red, slightly elevated papules, which are more or less scaly. An erythematous patch is gradually formed by the coalescence of these papules. The periphery of the patch is elevated and sharply defined, while the center is depressed and atrophied. The ducts of the sebaceous glands are dilated and often filled with sebum. The disease spreads very slowly, shows no tendency to ulceration, and rarely excites any subjective symptoms.

Diagnosis.—The location, the sharply defined red patch with an elevated margin and depressed center, the slight scaliness, the dilated sebaceous ducts, the chronic course, and the absence of ulceration are the diagnostic features.

Lupus Vulgaris.—This affection begins earlier in life, is characterized by tubercles and ulceration, and lacks involvement of the sebaceous glands.

Prognosis.—Favorable under prolonged and judicious treatment.

Treatment.—General tonics, like iron, arsenic, phosphorus, and cod-liver oil, are often indicated.

Local Treatment.—In many cases mild applications accomplish the most good. Much benefit is often derived from washing the part thoroughly with green soap and alcohol for a few days and then applying the following lotion:

℞. Zinci sulphatis
Potassii sulphidi . . . . āa ʒij
Aquæ . . . . f℥iij
Alcoholis . . . . f℥j.—M.
SIG.—Shake well, dab the parts for fifteen minutes twice daily, and allow to dry on. (DUHRING.)

In sluggish cases stimulating applications are useful. One like the following may be selected:

℞. Acidi pyrogallici . . . . ʒss–j
Petrolati . . . . aa ℥ss.—M.
SIG.—Apply locally. (KAPOSI.)

Treatment by the *x*-ray has been followed by excellent results in some very obstinate cases.

LUPUS VULGARIS.

(**Lupus Exedens.**)

Definition.—A local manifestation of tuberculosis, characterized by soft red tubercles that usually terminate in ulceration and scarring.

Etiology.—Early life and female sex are general predisposing factors. It is comparatively rare in this country, but very common in Austria and Germany. The exciting cause is the tubercle bacillus.

Symptoms.—Lupus vulgaris most frequently manifests itself on the face, especially near the nose. It begins as minute, deeply seated, reddish-brown papules which grow very slowly until they reach the dimensions of tubercles. They are smooth, quite soft, and seldom painful. At this stage they may either undergo slow absorption or, which is more frequent, break down and leave chronic ulcers. The ulcers are shallow, and their edges are soft and red. There is very little discharge. They spread slowly, and may involve all the soft parts, but the bone is never invaded. While one part of the ulcer is spreading, other parts are being filled with shriveled cicatricial tissue which in turn is often the seat of new tuberculous nodules.

Diagnosis.—**Epithelioma.**—Epithelioma is a disease of advanced life; it begins as a firm, wax-like nodule; the resulting ulcer starts from a single point; its borders are distinctly elevated and hard; it secretes a blood-streaked fluid; and it is often painful.

Syphilis.—The age, history, associated evidences of syphilis, the rapid course, the deep ulcers, the abundant offensive discharge, and later the involvement of the bones, are the diagnostic features.

Prognosis.—Very guarded. Its removal is often followed by relapse.

Treatment.—General tonics, like iron, arsenic, phosphorus, and cod-liver oil, are usually indicated.

Local Treatment.—The growth may be removed by cauterization, cureting, excision, or electrolysis. One of the following caustic applications may be employed:

℞.	Acidi salicylici	ʒss–j
	Collodii	f℥j.—M.

Or:

℞.	Acidi pyrogallici	ʒij–iij
	Vaselini cerati resinæ . . . āā q. s. ad	℥j.—M.

(STELWAGON.)

Often the best results are obtained by cureting and subsequently applying caustics.

Both phototherapy and *x*-ray therapy have also been used with considerable success in the treatment of lupus.

SYPHILIS CUTANEA.

The *secondary symptoms* appear between the first and the fourth month following the chancre, and are characterized by a symmetric arrangement, a coppery color, polymorphism (many forms at the same time), and an absence of itching. They are usually associated with certain general symptoms, such as sore throat, pain in the bones, loss of hair, enlargement of the lymphatic glands, and failure of health.

The *tertiary symptoms* appear in from six months to several years after the primary sore. They are, as a rule, localized, are tubercular, gummatous, or ulcerative in form, and tend to group.

Macular Syphiloderm.—This is a secondary manifestation, and consists in a general eruption of dark-red macules, varying in size from a millet-seed to a ten-cent piece.

Diagnosis.—*Measles.*—The absence of fever, of catarrh, of a crescentic arrangement, together with the history, will prevent an error in diagnosis.

Papular Syphiloderm.—This may be an early or late manifestation, and is characterized by a general eruption of large or small, dull-red papules. A few pustules are also frequently present. It pursues a chronic course, finally disappearing by desquamation, and leaving behind slight pigmentation.

Diagnosis.—The history, distribution, dark color, and the presence of pustules will separate it from keratosis pilaris, papular eczema, and lichen ruber.

Tuberculous Syphiloderm.—A late manifestation, characterized by a localized eruption of dark-red, shiny papules varying in size from a pea to a large bean. By some these tubercles are regarded as gummatous in character. They pursue a chronic course and finally disappear by absorption or ulceration. The ulcers thus formed, when single, are round, punched out, and frequently covered with crusts; when they coalesce, they form a serpiginous sore that pours forth a thick yellowish discharge.

Diagnosis.—*Lupus Vulgaris.*—This occurs in earlier life; it pursues an extremely chronic course; the ulcer is superficial; the tubercles are soft, and frequently redevelop in the scar tissue; the secretion is scant; and the bone is never involved.

Epithelioma.—In this affection the progress is slower; there is only one point of ulceration; the secretion is scanty; and the border is markedly infiltrated.

Bullous Syphiloderm.—This is a late manifestation, and is characterized by an eruption of well-filled blebs varying in size from a coffee-bean to a walnut. The contents of the blebs are puriform. They subsequently form dark, conic, stratified crusts under which are ulcers pouring forth a thick, purulent fluid.

Diagnosis.—*Pemphigus.*—The history, the concomitant symptoms of syphilis, and thick, greenish crusts will serve to distinguish syphilis from pemphigus.

Gummatous Syphiloderm.—This appears as a firm, circumscribed nodule that gradually turns red and softens. It may disappear by absorption, or break down and leave a deep, punched-out ulcer.

Moist Papules (Mucous Patches).—These consist in soft flat papules covered with an offensive, grayish secretion. Heat and moisture favor their development, so that their favorite seats are around the arms, the genitalia, the mouth, and in women under the mammæ.

Papulosquamous Syphiloderm.—This may be an early or late manifestation, and is characterized by a general eruption of small papules that are more or less scaly, so as to resemble psoriasis.

Diagnosis.—The history, the slight scaling, the dirty-gray color of the scales, the dark-red color of the lesions, the especial tendency to involve the palms and soles, will serve to distinguish syphilis from *psoriasis.*

Squamous Eczema.—In this affection the distribution, the infiltration of the skin, and the marked itching will lead to a correct diagnosis.

Annular Syphiloderm.—In this form the lesions consist of circles or semicircles of small, dark-red papules.

Pustular Syphiloderm.—This form usually appears within the first year, and is characterized by a general eruption of small or large, acuminated or flat pustules that finally dry up and form yellowish-brown crusts. Large lesions leave superficial ulcers. The term *rupia* is applied to large, conic, stratified crusts that rest loosely on the ulcerating basis.

Diagnosis.—*Variola.*—Absence of syphilitic history, the shot-like feel, the umbilication, the itching, the high fever, and the acute course will separate variola from syphilis.

Acne.—This is usually limited to the face and shoulders; there is no history of syphilis or concomitant symptoms of that affection.

Treatment.—The internal treatment consists in the administration of iodid of potassium, mercurials, and tonics.

℞. Hydrargyri iodidi . . . . gr. j
Potassii iodidi . . . . ʒiv
Syrupi sarsaparillæ compositi
Aquæ . . . . aa f℥ij.—M.
SIG.—Teaspoonful three times a day after meals.
(R. W. TAYLOR.)

Or:

℞. Hydrargyri protiodidi . . . . gr. v–x
Extracti opii . . . . gr. iv.—M.
Fiant pilulæ No. xx.
SIG.—One morning and evening. (HARDAWAY.)

Local Treatment.—Papular eruptions may be washed with mercurial lotions; mucous patches may be dusted with calomel; ulcers may be dressed with iodoform.

LEPROSY.

(Lepra; Elephantiasis Græcorum.)

Definition.—A chronic contagious disease, excited by the bacillus of leprosy, and characterized by tubercular formations, ulcerations, atrophy, disturbances of sensation, and an increase or decrease of pigment.

Etiology.—The disease is contagious, but direct inoculation is essential to its transmission. It seems to be more common in hot climates. The exciting cause is the Bacillus lepræ, which closely resembles the tubercle bacillus.

Varieties.—There are two varieties: tubercular leprosy and anesthetic leprosy; but the two forms are often associated in the same patient.

Symptoms.—Certain prodromes may precede the outbreak of the disease, such as malaise, headache, chilliness, depression of spirits, and numbness in the parts to be affected.

Tubercular Leprosy.—In this form spots of erythema appear on the body; they soon become pigmented and hyperesthetic, and develop into tubercles varying in size from a pea to a walnut. The face, extremities, and genitals are the parts most commonly affected, but occasionally the mucous membranes, especially of the nose and throat, are invaded. Ultimately the tubercles may break down and leave superficial indolent ulcers. In some cases a bullous eruption appears from time to time. The hair, eyebrows, and eyelashes fall out, the eyes become inflamed, the features distorted, and the voice husky. The disease may last many years, death finally resulting from exhaustion or some intercurrent disease.

Anesthetic Leprosy.—In this form the peripheral nerves are invaded by the Bacillus lepræ. The outbreak may be preceded by numbness, itching, or lancinating pains. These symptoms are followed by the appearance of discolored spots, which are at first associated with hyperesthesia, but later more or less anesthesia develops. The skin and its appendages atrophy, the bones undergo necrosis, and the phalanges drop off one by one. In some cases (lepra alba) the skin is not only anesthetic, but distinctly white. Finally, when the nerves are more or less destroyed, paralysis results. The duration is many years.

Prognosis.—Unfavorable. A cure is practically impossible, though the progress of the disease may be stayed by appropriate treatment.

Treatment.—Sufferers should be isolated. Tonics are usually indicated. Chaulmoogra oil and gurjun oil, internally and externally, have been highly recommended. Externally, chrysarobin, ichthyol, or resorcin may be applied to the affected parts.

EPITHELIOMA.

(Skin Cancer.)

Etiology.—Late life, heredity, and local irritation are the predisposing factors.

Varieties.—Superficial, deep-seated, and papillomatous.

Superficial Epithelioma (Rodent Ulcer).—This form usually begins as a firm, circumscribed, reddish-yellow, wax-like papule. After the lapse of several months or years the papule becomes scaly, and the removal of the scales is followed by a slight excoriation, which in turn becomes covered with a slight, reddish-brown crust. The latter tends to adhere, and its repeated removal is followed by a raw surface, which is gradually converted into an ulcer. The ulcer has a prominent indurated margin ; its outline is irregular ; its base is uneven and glazed ; and it exudes a sanious, viscid excretion. It is not painful ; it does not lead to enlargement of the neighboring lymphatic glands ; nor does it cause impairment of the general health. It spreads very slowly, and sometimes becomes stationary or actually heals. More frequently the ulceration continues until it involves all the tissues of the part, even the bones. The ulcer generally appears on the face, and in its advance it may destroy the nose, eyes, or a large portion of the cranial bones.

Deep-seated Epithelioma.—This variety may begin as a deep-seated, red, shiny tubercle, or it may develop from the superficial form. The ulcer which is ultimately formed is deep; its base is granular; its edges are everted, indurated, and of a reddish-purple color; it secretes a blood-stained yellow fluid; it is the seat of lancinating pain; it causes enlargement of the neighboring glands; and it sooner or later induces the cancerous cachexia. Death may result from exhaustion, or, more rarely, from hemorrhage caused by ulceration of a large blood-vessel.

Papillomatous Epithelioma.—This may begin as a warty excrescence, or may develop from one of the preceding varieties. It is characterized by an ulcerated surface from which springs an aggregation of large, highly vascular papillæ. Between the papillæ there are often deep-seated fissures from which exudes an offensive viscid discharge. The general health is impaired and the neighboring glands are enlarged.

Diagnosis.—**Lupus Vulgaris.**—Lupus begins in the young; the original papule is soft; there is often more than one center of ulceration; the margins of the ulcer are not hard and everted; the progress is extremely slow; the discharge from the ulcer is very scant, and the bones are never involved.

Syphilis.—The history, the associated evidences of syphilis, the rapid progress of the ulceration, the abundant discharge, the absence of pain, and the effect of treatment will suggest the diagnosis.

Prognosis.—Guarded. A thorough removal in the beginning of the disease is often followed by a permanent cure. When the process is advanced, the growth usually returns.

Treatment.—Epitheliomatous growths may be removed by the use of caustics, the cautery, the curet, or by excision. The last is preferable when the growth is small and circumscribed.

Phototherapy and *x*-ray therapy have recently been employed with considerable success.

AINHUM.

Ainhum is a rare affection, occurring chiefly in the colored race, and characterized by the appearance of a groove or furrow at the base of one or more of the toes. The groove deepens, the affected member becomes swollen, and finally drops off at the point of strangulation.

DERMATALGIA.

Dermatalgia, or neuralgia of the skin, is a rare affection, and is characterized by paroxysms of sharp, lancinating pain in the skin, which arise without any change in the local appearance. It is most frequently observed in women of a neuropathic tendency, and may arise from any of the causes which induce neuralgia elsewhere.

Treatment.—The cause must be sought for and, if possible, removed. Tonics, like iron, arsenic, quinin, and phosphorus, are often indicated. Locally, massage and electricity may prove useful.

PRURITUS.

Definition.—Pruritus is a functional affection, characterized by itching which is unassociated with any objective phenomena.

Etiology.—Pruritus may arise without obvious cause, as the *pruritus senilis* observed in the old, and the *pruritus hiemalis* which develops on the approach of cold weather and disappears when the weather becomes warm.

Symptomatic Pruritus.—Pruritus may be a symptom of many conditions, notably diabetes, gout, lithemia, hysteria, neurasthenia, and Bright's disease.

Symptoms.—There is only one symptom, and that is itching; but as a result of scratching, the part may become hyperemic, thickened, or the seat of eczema.

Diagnosis.—Pruritus must be distinguished from the itching induced by *pediculosis, or some local disease, like eczema.*

Prognosis.—This will depend on the cause. When the primary disease is curable, the prognosis for permanent relief is favorable. In other cases temporary relief only is to be expected.

Treatment.—Search should be made for the exciting cause, which should be removed, if possible. In all cases the urine must be examined for sugar, since diabetes is one of the most frequent causes of pruritus. Among the internal remedies recommended for pruritus may be mentioned nux vomica, belladonna, and pilocarpin. The best local remedies are carbolic acid, vinegar, thymol, chloral-camphor, boric acid, resorcin, menthol, hydrocyanic acid, and hot water.

℞.	Resorcinolis	gr. xv–xxx
	Sodii chloridi	gr. xv
	Glycerini	f℥ij
	Liquoris calcis	q. s. ad f℥iv.—M.

(HARTZELL.)

℞.	Acidi carbolici	ʒj–iij
	Glycerini	f℥ij
	Alcoholis	f℥ij
	Aquæ	q. s. ad Oj.—M.

℞.	Acidi carbolici	gr. xv
	Hydrargyri chloridi mitis	gr. xx
	Unguenti zinci oxidi	℥j.

SIG.—Apply locally in pruritus ani.

TINEA TRICHOPHYTINA.

(Ringworm.)

Definition.—A contagious disease excited by a vegetable parasite—the trichophyton.

Varieties.—On the scalp it is termed *tinea tonsurans;* on the body, *tinea circinata;* on the bearded region, *tinea sycosis.*

TINEA TONSURANS.

This form is observed almost exclusively on the scalp of children. It is characterized by one or more rounded, scaly, elevated, grayish-colored patches through which project dry, brittle, lusterless, broken-off hairs.

Diagnosis.—**Seborrhea.**—The patches are not circumscribed; the scales are greasy; the hair is not involved; and the microscope reveals no parasite.

Eczema.—The patches are not circumscribed; the hair is not involved; there is more inflammation; there is marked itching; and the microscope reveals no parasite.

Alopecia Areata.—Baldness is complete; there are no scales; and the base is smooth and shiny.

Prognosis.—Favorable.

Treatment.—Tonics are often indicated. The parts should be thoroughly washed with soap and water, and the affected hairs removed. The following parasiticides may be employed in ointment or lotion; mercury, sulphur, chrysarobin, or sulphurous acid.

℞. Hydrargyri ammoniati	ʒj
Petrolati	℥j.

Sig.—Apply once or twice daily.

Or:

℞. Betanaphtholis	gr. xl
Sulphuris præcipitati	ʒj
Vaselini	℥j.—M.

Sig.—Rub into affected area once or twice daily.
(Hardaway.)

TINEA CIRCINATA.

(Ringworm of the Body.)

This appears as one or more rounded, red, slightly elevated scaly patches, which on close examination reveal minute vesicles or papules. As the disease advances new patches spring from the periphery, while the central portion clears up. There is often considerable itching.

Diagnosis.—**Psoriasis.**—The marked scaling; the absence of itching; the tendency to involve the extensor surfaces, especially the knees and elbows; and the absence of the trichophyton will separate psoriasis from ringworm.

Eczema.—The patches are ill defined; do not clear in the center; there is more infiltration of the skin; and there is no trichophyton.

Prognosis.—Favorable.

Treatment.—Tonics are frequently indicated; mercury, sulphur, sulphurous acid, and hyposulphite of sodium are among the best parasiticides.

℞. Sodii hyposulphitis	ʒij
Aquæ	f℥ij.—M.

Sig.—Apply locally. (Duhring.)

Or:

℞. Hydrargyri ammoniati	gr. xxx
Adipis	℥j.—M.

Sig.—Apply locally.

TINEA SYCOSIS.

(Barber's Itch; Sycosis Parasitica.)

This begins as a red, scaly patch involving the bearded region. Soon purplish tubercles and pustules form around the opening of the hair-follicles, and the hairs become lusterless, brittle, and loose. There is often considerable itching.

Diagnosis.—**Simple Sycosis.**—In this the inflammation is superficial; the hairs are not involved; and the trichophyton is absent.

Eczema.—The tubercles, the involvement of the hairs, and the presence of the trichophyton will separate it from eczema.

Prognosis.—Favorable; unless treated actively, however, there may be a permanent loss of hair.

Treatment.—The affected hairs should be removed, and one of the following parasiticides employed in lotion or ointment: mercury, sulphur, or hyposulphite of sodium.

℞. Sodii hyposulphitis	ʒiij
Aquæ	f℥iij.—M.

SIG.—Apply locally.

Or:

℞. Sulphuris sublimati	ʒij
Vaselini	℥ij.—M.

SIG.—Apply locally.

TINEA VERSICOLOR.

(Pityriasis Versicolor.)

Definition.—A chronic affection excited by a vegetable parasite, the Microsporon furfur, and characterized by fawn-colored scaly patches which usually appear about the chest.

Etiology.—It is a disease of adult life, and is more frequently observed in the debilitated and uncleanly.

Symptoms.—It appears usually on the front of the chest as small round spots of a pale-yellow or fawn color, which slowly enlarge, fuse, and form slightly elevated, scaly patches. Subjective symptoms are generally absent.

Diagnosis.—**Chloasma** somewhat resembles tinea versicolor, but the former is not often observed on the trunk, is not scaly, and is not associated with a parasite.

Prognosis.—Favorable.

Treatment.—The parts should be frequently washed with soap and water, after which one of the following parasiticides may be applied: Corrosive sublimate (2–3 grains to an ounce of water), sulphurous acid, or hyposulphite of sodium:

℞.	Sodii hyposulphitis	℥v
	Glycerini	f℥iij
	Aquæ	q. s. ad f℥v.—M.

SIG.—Apply locally.

Or:

℞.	Hydrargyri chloridi corrosivi	℈j
	Alcoholis	f℥iv
	Saponis viridis	℥ij
	Olei lavandulæ	fʒj.—M.

SIG.—To be rubbed in well night and morning.

(VAN HARLINGEN.)

TINEA FAVOSA.

(Favus.)

Definition.—A contagious affection of the scalp excited by the Achorion Schönleinii, and characterized by yellowish, cup-shaped crusts.

Etiology.—It is observed especially in poor, ill-nourished children.

Symptoms.—The disease is characterized by one or more rounded, yellow, cup-shaped crusts, through which project dry, brittle, lusterless hairs. The underlying tissue is more or less atrophied and scarred. It is associated with some itching and a peculiar musty odor.

Diagnosis.—The yellow, cup-shaped crusts, the odor, and the atrophy of the skin will separate it from **ringworm.**

Prognosis.—Favorable. When not treated early, it may be followed by permanent baldness.

Treatment.—The crusts should be removed by oil or soap and water. The affected hairs should also be removed. The following parasiticides are efficient: mercury, sulphur, chrysarobin, and hyposulphite of sodium.

SCABIES.

(Itch.)

Definition.—Scabies is a contagious disease excited by an animal parasite—the Acarus scabiei—and manifested by papules, vesicles, pustules, burrows, and intense itching.

Etiology.—The disease is always acquired through intimate intercourse with patients already affected.

Symptoms.—The disease manifests itself by intense itching, which is associated with an eruption of small papules, vesicles, and pustules. Among these lesions may be found cuniculi, or burrows; these are discolored, dotted, slightly elevated lines, ranging from a line to half an inch in length, and produced by the penetration of the female acarus and the deposition of her eggs along the passage. The parts most commonly affected are the hands, between the fingers, the wrists, the axillæ, the genitalia, beneath the mammæ, and the inner aspects of the thighs. The face and scalp are never involved.

Diagnosis.—The recognition of scabies rests on the history, the itching, the presence of burrows, the multiformity of the lesions, and their peculiar distribution.

Prognosis.—Favorable.

Treatment.—Ointments of sulphur, styrax, and naphthol are efficient remedies. After a thorough bath the whole body should be anointed twice daily for three or four days. At the end of this time the bath should be repeated, and the bed-linen and underclothing changed. The infected clothing should be sterilized.

℞.	Sulphuris sublimati	ʒj
	Balsami Peruviani	ʒss
	Adipis	℥j.—M.

SIG.—Rub in thoroughly twice daily. (DUHRING.)

℞.	Balsami styracis	ʒiv
	Adipis	℥iss.—M.

PEDICULOSIS.

(Phtheiriasis.)

Pediculosis Capitis.—This form results from the pediculus capitis, or head-louse, a gray insect from one to two millimeters in length. The condition is recognized by itching of the scalp and the discovery of the lice or their white ova, or nits. Eczematous lesions resulting from scratching are often observed.

Pediculosis Corporis.—This form results from the pediculus corporis, pediculus vestimenti, or body-louse, a somewhat larger insect than the head-louse. The condition is recognized by intense itching on the covered parts of the body, scratch-marks, petechiæ caused by the bite of the insect, and the discovery of the lice on the garments.

Pediculosis Pubis.—This form results from the pediculus pubis, or crab-louse, a minute, gray, translucent insect. It is found on parts covered with short hair, as the pubes, axillæ, eyebrows, etc.

Treatment.—In *pediculosis capitis* the head may be thoroughly washed with coal-oil, dilute carbolic acid (1 dram to 1 pint), or tincture of cocculus indicus.

In *pediculosis corporis* the parts should be thoroughly washed and the clothes subjected to a high temperature. The body may be bathed in a weak solution of corrosive sublimate.

In *pediculosis pubis* a lotion of corrosive sublimate (2 grains to 1 ounce) or an ointment of ammoniated mercury (1 dram to 1 ounce) is very efficient.

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